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THE UNIMPORTANCE OF COTTON SEED IN THE DISSEMINATION OF VERTICILLIUM WILT IN CALIFORNIA[1]

B. A. RUDOLPH[2] AND G. J. HARRISON[3]

(Accepted for publication March 30, 1944)

Verticillium wilt of cotton was first reported in California by Shapovalov and Rudolph in 1930 (11) following the isolation of the causal fungus (*Verticillium albo-atrum* R. and B.) from diseased plants grown at the U. S. Cotton Field Station at Shafter. Herbert and Hubbard (6) claim to have observed the disease in 1927, which is probably true, since the symptoms are very characteristic and not likely to be confused with those of other cotton diseases in California. Today Verticillium wilt is recognized as a destructive disease in virtually all cotton-producing sections of the San Joaquin Valley. In some fields the incidence of infection is virtually 100 per cent, and much of the cotton harvested from such fields is of poor quality.[4]

Because of the seemingly rapid spread of Verticillium wilt throughout all cotton districts of the State in comparatively few years, it was only natural that growers and plant pathologists alike should suspect seed transmission of the parasite. Investigators in Texas and Arizona, where this disease has spread with equally spectacular rapidity, have already reported seed transmission of wilt. Unfortunately, there is no agreement as to how, when, or where the seed becomes infected or contaminated by the fungus. Thus, Taubenhaus, in Texas, reported isolating *Verticillium* from the interior of 8.3 per cent of 1440 seeds in 1936 (12) and from 2.3 per cent of 1600 seeds in 1937 (13). In a personal letter to the senior writer, Taubenhaus particularly stressed the point that the fungus inhabited the interior of seeds and not the lint. He not only delinted the seed with sulphuric acid but also disinfected it with a mercuric solution. He said that no recommendations were being made (presumably to growers) until the work could be replicated 2 or 3 years. He pointed out that "one year the infection in the interior of the delinted seed is present and another year it may not be."

In contradistinction to the results obtained by Taubenhaus are those of Brown[5] (1) in Arizona. He was unable to isolate the fungus from the interior of the seed but claims to have succeeded in isolating it from the lint. He has given no details of his experiments, and no one else has made such claim. Brown (2) has reported instances of the disease appearing in

[1] Much of the work reported in this paper was done under W.P.A. Projects 50–1859, 50–11992, and 50–12456–D.

[2] Associate Plant Pathologist in the California Agricultural Experiment Station.

[3] Agronomist, United States Department of Agriculture.

[4] In addition to ripening prematurely, the bolls on affected plants are stunted, and the lint is of inferior quality as to fiber length and strength. Studies at the U. S. Cotton Field Station at Shafter confirm these observations.

[5] Although this report is not signed, it and similar articles that appeared in Arizona newspapers, such as the Arizona Star (Tucson), released to the press by the University of Arizona, credit Dr. Brown and his associates with the work.

clean fields planted to seed that had not been delinted, but he knew of no instance where the disease had developed in clean fields planted to acid-delinted seed. Presley,[6] on the other hand, has reported the appearance of the disease in desert areas of Arizona, cropped for the first time and planted with acid-delinted seed.

In letters to Miles (8, 9), Sarejanni has expressed the belief that Verticillium wilt is seed-borne, but he was unable to demonstrate the presence of the fungus in seed produced on diseased plants. Sarejanni first observed the disease near Copias, Greece, in September, 1932, in cotton plants grown from seed imported from North Carolina that same year. Seed obtained in 1932 from Virginia produced healthy plants, but it is not certain that the seed purchased in Virginia actually was grown there. The disease has never been reported or recognized in either of those States, according to letters received in November, 1942, from pathologists of the North Carolina and Virginia Agricultural Experiment Stations.

Miles (8) likewise was unable to demonstrate the presence of the parasite within cotton seed.

SEED STUDIES IN CALIFORNIA

Experiments to determine whether *Verticillium albo-atrum* is transmitted by cotton seed and whether infected seed is an important factor in California, were made from 1934 through 1938.

Each year mature cotton plants in the most advanced stages of Verticillium wilt were dug at intervals at the United States Cotton Field Station at Shafter, California, where the writers are collaborating in the production of a Verticillium wilt-resistant cotton suitable to California. Cultures of the diseased plants were made at the University of California Deciduous Fruit Field Station at San Jose, California. The plants grown at Shafter had been artificially infected by injecting pure cultures of the fungus into the soil about the roots.

Internal Seed Studies. In 1934, to determine whether the fungus is capable of infecting the internal structure of the seed by way of the vascular system, sections of the stele of each plant near the roots were cultured on Czapeck's nutrient agar, slightly acidified with lactic acid to suppress bacterial growth. At the same time, varying numbers of mature, unopened bolls, particularly those on the lowest branches, close to the main stalk, were clipped off and each put in a paper bag and kept in a dry place at room temperature.

Pending the outcome of the cultures made from the steles, Petri-plate cultures were made from either the receptacle or placentae of each boll to determine whether they were diseased. Bracts were completely removed, then the receptacle was sliced off close to the base of the boll. Next, the

[6] Presley, John T. The occurrence of Verticillium albo-atrum wilt on cotton grown as a first crop in certain remote desert areas. Unpublished manuscript.

This paper was read before the Pathological Section of the Association of Southern Agricultural Workers at Memphis, Tenn., 1942. References to and quotations from it have been made with the author's consent.

receptacle and its short peduncle were scrubbed with a brush and lightly scraped with a scalpel to remove any dirt or dead cuticle adhering to them. After a final rinsing with tap water the preparation was surface-sterilized in mercuric bichloride solution 1/1000 for 3 minutes, rinsed with sterile, distilled water, and cut into at least 3 pieces, the usual precautions to insure asepsis being taken at all times. Plantings were made in Petri plates of slightly acidified Czapeck's nutrient agar. A plate never was discarded in less than 3 weeks from the time the cultures were made, unless the fungus could be identified earlier, microscopically.

Cultures were made from the placentae of many bolls as follows: The bracts and receptacle of each boll were cut away and discarded. The carpels of the mature, unopened bolls were cut apart with a knife drawn along the sutures and worked free from the unexpanded locks by hand. The locks, after properly identified, were placed in paper bags and kept in a dry place at room temperature, pending the results of the cultures. The axile and numerous placentae of cotton form a central column that can easily be broken into its component parts with the fingers. Before doing this, however, the numerous tough, parchment-like walls that are attached to the placental column and divide the boll into locules were cut away. The individual placentae, from which the locular wall tissue had been trimmed away, were surface-sterilized in mercuric-bichloride solution 1/1000 for 3 minutes, after which they were rinsed in sterile distilled water and planted in Petri plates of slightly acidified Czapeck's nutrient agar. Figure 1 shows the successive steps taken in preparing both the receptacle and placentae for culture purposes.

Finally, cultures were made from the seed of 28 of the bolls, the receptacles of which had yielded *Verticillium* in culture. The seed was delinted either with sulphuric acid or by scraping it with a scalpel until all traces of fiber were removed. After rinsing in tap water, it was surface-sterilized by immersion in mercuric-bichloride solution 1/1000 for 3 minutes, then was rinsed with sterile distilled water. Each seed was then cut transversally into its micropilar and funicular halves which were planted in Petri plates of slightly acidified Czapeck's nutrient agar. To maintain the identity of each seed, the halves were planted in proper sequence on either side of a line drawn across the bottom of the plate with a wax pencil, the funicular halves on one side and the micropilar on the other. Not more than 4 seeds (8 halves) were planted to a plate. None of the 763 seeds cultured yielded *Verticillium*.

The results obtained in 1934 are summed up in table 1.

To determine what other parts of the boll might become infected, cultures were made from the carpels of 5 bolls, of which either the receptacles or placentae had yielded *Verticillium* in culture. The tissue was cut into fragments approximately one-half inch square, washed, and surface-sterilized as described. The tissue was found to be sterile.

Cultures also were made from the bracts of 14 bolls selected from 5 plants that had been proved to be diseased by cultural methods. The fungus had

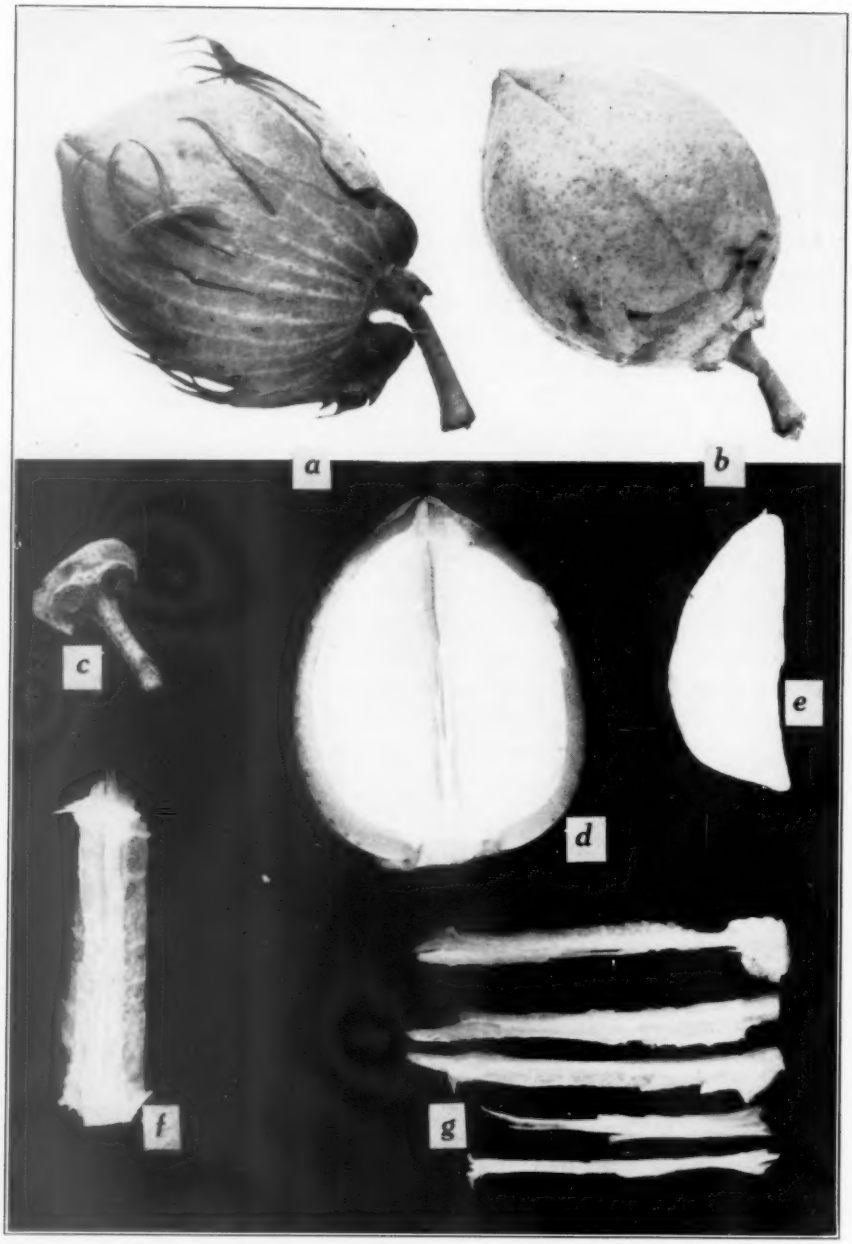

FIG. 1. Acala cotton boll and its component parts. Natural size. a. A mature boll with leafy bracts. b. A mature boll with bracts removed. c. The receptacle, trimmed and ready for sectioning and culturing. d. Longitudinal section through a boll showing the central placental column and core at its base. e. Individual lock of mature, moist, unexpanded cotton. f. Central placental column with parchment-like membranes separating the locks cut away. g. The placentae, which make up the central placental column, separated. Note core attached to the base of the top placenta. The second and third placentae are in position to show the tooth-like protuberances to which the seeds were attached.

TABLE 1.—*Results of cultures made in 1934 from either the receptacles or placentae, as well as the seed of Acala cotton plants severely affected with Verticillium wilt*

No. diseased plants	23
No. bolls from diseased plants cultured	139
No. bolls yielding no *Verticillium* from either receptacles or placentae	98
No. bolls with healthy placentae	71
No. bolls with healthy receptacles	27
No. bolls yielding *Verticillium* in culture	41
No. bolls with diseased placentae	1
No. bolls with diseased receptacles	40
No. seed cultured[a]	763
No. healthy seed	763
No. diseased seed	0

[a] From 28 bolls only, including the one with diseased placentae. Time did not permit culturing seed of the remaining 13 bolls with diseased receptacles.

reached the receptacles of 13 of these bolls. Of the latter, 3 had diseased bracts. Although the receptacles of the 14th boll failed to yield the fungus in culture, the bracts gave positive evidence of its presence. Recently dried or green bract tissue yielded the fungus in equal proportions.

In 1935 and 1936 cultures invariably were made from both the receptacle and placentae of each boll, to determine the ratio between placental infections and receptacle infections. The seed was delinted with sulphuric acid. Only seed from bolls with diseased receptacles or placentae were cultured (Table 2).

Since the accumulated data showed that the fungus only occasionally reaches the receptacles, in 1937 and 1938 cultures were made first from the receptacles. If a receptacle culture was positive, cultures were then made from the placentae of the same boll. This made it possible to work with a much greater number of bolls (Table 2).

External Seed Studies. Several hundred cultures were made from Acala cotton seed deliberately selected from the bottom of pickers' bags where the lint and fuzz were most likely to become contaminated with soil-

TABLE 2.—*Results of cultures made 1935–1938, inclusive, from both the receptacles and placentae, as well as the seed, of Acala cotton plants severely affected with Verticillium wilt*

Year	No. of diseased plants	No. of bolls cultured	No. bolls with diseased receptacles	No. bolls with diseased placentae	No. of seed cultured	No. of diseased seed
1935	12	61	1	0	?[a]	0
1936	53	434	7	1	144[b]	0
1937	101	671	2	0	34[c]	0
1938	137	2066	100	0	2961	0
Totals	303	3232	110	1	3139	0

[a] The seed count of this single boll was overlooked when the plates were discarded. Cultures made from the seed were sterile.

[b] From only six of the seven bolls with diseased receptacles. The seed from the single boll with diseased placentae was accidentally discarded before cultures of it were made.

[c] From only one of the two bolls with diseased receptacles. The other boll was badly decayed by *Alternaria* sp. while waiting the outcome of the receptacle cultures.

inhabiting organisms, without treatment of any kind. The plants that produced the seed were severely affected with the wilt, having been grown in soil artificially infested with the fungus.

The fiber on the seed was dusty, but the great bulk of plant débris had been removed in the ginning. Usually, only one seed was planted to a plate of slightly acidified Czapeck's nutrient agar. *Verticillium* failed to develop, but other fungi, too numerous to mention, quickly overran the plates.

MICROSCLEROTIA

Verticillium albo-atrum occasionally produces microsclerotia on old raspberry canes (4, 5, 10), apricot (3), and prune twigs (10). How long those that find their way to the soil remain viable is not known. At the Deciduous Fruit Field Station they have withstood desiccation in culture tubes for 10 years. Produced in any considerable quantity on old, dead cotton stalks left in the field to overwinter, it is conceivable that eventually such a large population of them might build up in the soil that contamination of the lint in pickers' bags dragged across the ground would be practically inevitable. Seed with lint thus contaminated might disseminate the fungus.

To determine whether *Verticillium albo-atrum* produces microsclerotia on dead cotton plants, experiments were started in 1937 and concluded in 1942. Each year, a patch of artificially infected plants that had developed the disease in severe form was left untouched throughout the winter at the United States Cotton Field Station at Shafter. Plants were dug and forwarded to the Deciduous Fruit Field Station at San Jose only when it became imperative to put the ground in order for spring planting.

The plants, including the main stalks and branches, were cut into convenient lengths of about 6 inches, and examined under the microscope for any kind of minute, black body that might possibly be a microsclerotium of the parasite. Representative samples of all such bodies, regardless of their origin, whether erumpent or wholly superficial, were picked up with needles and transferred to Petri plates of Czapeck's nutrient agar, some of which were acidified and some not.

Prior to making the cultures the twigs were either left untreated or were surface-sterilized by immersion in mercuric bichloride solution 1/1000 for periods ranging from 1½ to 3 minutes, and then rinsed with sterile, distilled water. The number of plantings per plate varied from 5 to 8. A total of 11,723 cultures was made during the 5-year period. Neither *Verticillium albo-atrum* nor any of its closely related forms developed in any of the cultures made.

LONGEVITY OF THE FUNGUS IN DEAD COTTON STALKS

Each year many cultures were made from the wood of the old plants used in the microsclerotia studies, to determine to what extent the parasite lives over in the main stem and twigs. Only infrequently could the fungus

be cultured, which indicates that it dies fairly rapidly in the tops of the plants, probably after they have been killed by frost. The fungus could be cultured fairly easily, however, from the heavier woody portions of the main stem at ground level and from the tap root in which decay was not too far advanced.

COTTON FIBER INOCULATIONS

To determine whether *Verticillium* can attack mature cotton fiber, either dry or wet, inoculations were made as follows: Locks of dry cotton were removed from the bolls and seeded by hand. The fiber was then fairly loosely packed in standard culture tubes, to some of which distilled water had been added in varying amounts. The tubes were plugged, steam sterilized in an autoclave, and inoculated with large fragments of sclerotial crusts of *Verticillium albo-atrum* lifted from the surfaces of cultures grown on Czapeck's nutrient agar. Such fragments simulated the occasional bits of dried bract or carpel tissue that occasionally cling to the seed after ginning, and probably even surpassed them in infectiousness.

Only cotton that was wet almost to the point of complete saturation was attacked. It turned a dark-grey, due to the presence of myriads of individual, black microsclerotia clinging to the fibers. The latter were not perceptibly discolored. The fungus failed to grow on the cotton in tubes to which no water had been added, and did not grow above the water line in those containing only small amounts of water. Presumably the humidity in such tubes was relatively high, but insufficient to enable the fungus to grow.

DISCUSSION

The considerable data obtained contribute nothing to support the theory that cotton seed from plants affected with Verticillium wilt may harbor the parasite internally. During the 5-year period of the experiments, a total of 3371 bolls from 326 plants, experimentally proved to be severely affected by the disease, were cultured. The fungus had reached the receptacles of only 150 of these bolls, and of the latter, only 2 showed infection of the core at the base of the placental column (Tables 1 and 2; Fig. 1, d and g). In figure 1, g, the core-like tissue referred to is shown attached to the first placental strip. The fungus grew only from this core-like tissue and never from the true placentae where the seeds were attached.

The original point of attachment between the funiculus and placenta is characterized by a tooth-like structure on the latter. The second and third placentae from the top in figure 1, g, clearly show these tooth-like points of seed attachment. Considering the fact that the vascular system is highly developed at these points it is not unreasonable to believe that had the fungus reached them it would have grown out and established colonies in the agar. Such was not the case.

The seed from the single boll, penetrated by the parasite in 1936 (Table 2), we regret to state, was accidentally discarded before cultures were made

of it. It is virtually certain that the seed was not infected, since the fungus never penetrated beyond the core at the base of the placentae. The seed from the boll, penetrated by the fungus in 1934 (Table 1) was cut in halves and cultured. It was not infected.

The average number of fully developed, viable seed per boll in healthy Acala cotton in California is 40. That of bolls from plants severely attacked by Verticillium wilt averaged only 32, the bolls being puny and stunted as a result of the disease. The 3371 bolls cultured, therefore, had a possible content of 107,872 seeds, all of them apparently free from Verticillium infection, since the experiments were so conducted that a check of the progress of the fungus in the stem, receptacle, and boll itself was possible. Actually, a total of 3902 seeds from bolls with infected receptacles were cut in half and cultured. All were healthy.

The late Dr. Taubenhaus (11, 12) probably is the only investigator to claim isolation of *Verticillium* from the inner tissue of cotton seed. Neither of his brief reports gives the details of his experiments. Yet he, himself, in personal correspondence with the senior writer, expressed doubt as to his findings. Pending further studies he preferred to make no recommendations to growers in Texas.

The writers do not wish to infer that infection of the inner tissue of cotton seed is impossible; but in view of accumulated negative data, it is improbable. It is conceivable that, under conditions ideal for the fungus, it may succeed in penetrating the inner tissue of an occasional seed, but this fact would have no practical significance. In the face of existing evidence other means of dissemination assume far greater importance.

Finally there is no assurance that the parasite having reached an occasional seed would remain alive there and attack the seedling the following spring. Such parasitized seed might not even germinate. It is conceivable that the fungus might be introduced into the soil with such seed. But, in the strict sense, it is wrong to assume that the disease is seed-borne without first planting such infected seed in healthful soil and observing whether it produces diseased plants.

The experiments to isolate the fungus from the fiber and fuzz left on the seed after ginning were discontinued primarily because of the nature of the disease itself and the basic habits of the parasite. Regardless of severity of wilt in the plant, the cotton in the boll is never attacked. It is true, the bolls ripen prematurely in diseased plants and are stunted and puny, but the lint is never decayed or even discolored. Since the lint is produced on the outside of the seeds, and the latter do not harbor the parasite, then contamination of the lint from outside sources could be the only source of danger. Logically spores or microsclerotia might be suspected of contaminating or even attacking the lint. Brown thinks this is possible, according to a letter to the senior writer.

Microsclerotia were not produced on the old, dead plants within a year; and even if plants were left in the fields a second year, it is doubtful whether

production of microsclerotia on the stalks would reach significant amounts in view of the comparative rapidity with which the fungus dies in the old wood. Therefore, the danger of the lint becoming contaminated with microsclerotia as the pickers drag their bags over the ground would seem negligible.

Verticillium albo-atrum, which is now known to attack over 200 widely unrelated hosts, sporulates readily in the saturated atmosphere of a culture tube, but rarely, if ever, in fields in California. There are virtually no references to spore production on field-grown plants in literature on the disease, and only occasional references to spore production on dying plants in hot frames. Van der Lek (7) describes the appearance of spores as "a very fine dew" on the necrosed leaf areas of cucumber plants growing in hot frames. Rudolph (10) also cites the comparatively few workers who have referred to spore production within the ducts and mesophyll of affected plants. Spore production on cotton plants in the field, in California at least, can be dismissed as of no practical importance. Therefore, the possibility of lint becoming spore-contaminated at the gin is very unlikely.

There still remains the possibility of the lint becoming infected from, or contaminated with, débris from the diseased plants themselves. Ginned seed usually has very little or no adhering débris, that most likely to be present being fragments of dried bracts. The fungus may invade the bracts, but how long it would remain alive in tiny, dry fragments of this tissue is unknown. Considering the comparative rapidity with which it dies in the woody stems, its chances for survival would seem to be slender.

In any case, the cotton fiber is a poor medium for the growth of the fungus. Only cotton wet to the point of saturation was attacked. Cotton, delivered to gins, ordinarily is sufficiently protected against rain or other moisture to preclude Verticillium infection of the fiber.

Herbert and Hubbard (6) harvested seed from diseased cotton plants and planted it without delinting it. The plants produced were healthy and normal in every way. The writers also repeated this experiment but were not certain that the ground in which they planted their seed was absolutely healthful. A small amount of disease developed under conditions that tended to confirm their suspicions that the soil was not sanitary at the outset.

Again, the writers do not wish to infer that transmission of Verticillium wilt by lint-bearing seed is wholly impossible, but rather that it is improbable. Among many other really important means of disseminating the disease, the use of such seed probably is of little or no importance.

Cotton-seed production in California is licensed. Production is confined as far as possible to healthy ground. Accordingly, the great preponderance of plants are free of Verticillium wilt and the seed produced is healthy. Even if some plants in the fields develop the wilt, the total amount of seed produced by them would be negligible in comparison to the total production. In the light of the experimental results obtained, the number of seed from such fields that could actually transmit Verticillium wilt is infinitesimally small.

Among the commoner, authentic and definitely proved means of disseminating the parasite may be mentioned the transfer of infective débris from one field to another on plows, tractors, etc. Cotton growers often transfer farm machinery long distances by truck, and little or no effort is made to remove coarser roots or other plant material clinging to it. The use of cuttings from diseased plants as well as the planting of infected nursery stock, including fruit trees and ornamentals, is one of the commonest methods of dispersal. Potato-seed tubers often are diseased, but may not be discolored. A common fallacy is the belief that all infected tubers have a brown discoloration of the vascular system or at least a brown spot at the point of stolon attachment. Most of them do, but certainly not all.

It has been stated that the disease has appeared suddenly in desert areas that had never been under cultivation. The inhabitants of such regions probably plant home gardens, shade trees, etc., and introduce the parasite with their plants and trees. In addition to stone fruits, bush fruits, truck crops, field crops, and ornamentals, *Verticillium albo-atrum* attacks many of our common weeds. It is not altogether impossible, therefore, that the disease may exist in weeds in some of the desert land cultivated for the first time. Reports of sudden "outbreaks" of the disease in virgin, desert soil are not wholly convincing. The average grower would not recognize or be concerned with the single, individual cotton plant that might develop the disease among the thousands in his field, assuming the disease to have been introduced on débris, etc. On the contrary, he would ignore it and eventually disk it into the soil along with the others at the end of the season, thereby establishing a real focus of infection the following year. The second year there might be enough disease present to constitute an "outbreak." The experiments and observations of Presley[7] in Arizona sustain the views expressed here. He states: "We have found that land in crop for the first time is often a veritable hot-bed of the disease and infections have appeared simultaneously in valleys which are widely separated and physiographically isolated. One of these areas known as Hidden Valley seemed to lend itself admirably to the study of *Verticillium* and its origin. The valley is well isolated from other cultivated areas, and pump water is used for irrigating the land. In 1937 Verticillium wilt was found there in a planting of Upland cotton, and though the disease was not severe several distinct spots were apparent. For three successive years additional land within a mile radius of the diseased field was brought under cultivation and planted to cotton as a first crop. Precautions were taken to obviate as far as possible any chance of seed-borne infection entering these plantings. The planting seed was acid-delinted at a commercial delinting plant in Mesa, Arizona, and one planting was made with seed of the D and P L variety that had been imported from an area known to be disease-free. In every instance the newly planted cotton showed, at various places in the field, typical wilt spots where the plants were killed outright or were severely stunted during early de-

[7] See footnote 6.

velopment producing bare spots in the field surrounded by plants which showed unmistakable symptoms of Verticillium hadromycosis."

SUMMARY

Cultures made over a 5-year period from 3371 mature cotton bolls produced on plants severely affected with Verticillium wilt showed that the fungus had succeeded in reaching only 150 receptacles (4.44 per cent), and had penetrated to the bases only of the placental columns of 2 bolls (0.00059 per cent). Cultures made from the seed produced by diseased plants showed it to be neither internally nor externally infected by *Verticillium*.

To test a theory that the lint on cotton seed might become contaminated with infective material existing in the ground as the pickers drag their bags over it, cultures were made from non-sterilized, lint-bearing seed taken from the bottoms of pickers' bags. The lint on such seed was dusty, but *Verticillium* never developed in the cultures. Similarly, to determine whether the parasite produces microsclerotia on dead cotton stalks left standing overwinter in a field, a total of 11,723 cultures were made during a 5-year period of the tiny, black bodies, erumpent or superficial, produced on such stalks. *Verticillium* was never found in the cultures. Also, to determine the longevity of the fungus in such old, dead stalks, a large number of cultures made over a period of years showed that the fungus dies out fairly rapidly in the tops after the plants have been killed by frost, but it remains alive until the following spring in many of the tap roots that have not decayed, as well as in the heavier woody portions of the stems at ground level. Lastly, to test a theory that the lint becomes infested by the fungus at the gins, cotton removed from bolls and steam-sterilized in culture tubes was inoculated with fragments of microsclerotial crusts of *Verticillium albo-atrum* produced on Czapeck's agar. The cotton constituted a poor medium; only lint wet practically to the point of saturation supported the growth of the fungus. It is exceedingly unlikely that cotton at the gins would become wet enough to enable *Verticillium* to attack it.

The experiments show that dissemination of Verticillium wilt by seed produced on plants affected by the disease is highly improbable. Also, that contamination or infection of the lint on healthy seed is equally improbable.

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INFECTION OF COTTON SEEDLINGS BY COLLETOTRICHUM GOSSYPII AS AFFECTED BY TEMPERATURE[1]

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Damping-off of cotton seedlings as caused by the anthracnose fungus was among the first of the seed-borne diseases to be studied intensively (1, 2, 3), yet there is little available information on the influence of specific environmental conditions on the incidence of infection by this fungus, except for the studies of Lehman (5). He studied the effect of 5 temperatures on the infection of seedlings which developed from seeds inoculated with spores of *Colletotrichum gossypii* South. Temperatures between 25° and 30° C. were most favorable for seedling infection. The data reported in this paper were secured in studies initiated to ascertain the extent to which Lehman's results might be applicable to naturally infested seed.

METHODS

Four lots of fuzzy cotton seed were germinated and the seedlings grown on 2 per cent agar gel, containing 0.002 *N* concentrations of KH_2PO_4, $Ca(NO_3)_2$, and $MgSO_4$, and having an initial pH of 6.5. Approximately 6-cc. portions were placed in 18 × 150-mm. test tubes, which were then plugged and autoclaved. The usual irregular germination of fuzzy cotton seed on an agar gel was overcome by liquefying the gel at the time of seeding, cooling it in a water-bath to 45° C., and then dropping the seeds on the liquefied gel. A small but sufficient amount of the fuzz on the seeds became embedded in the gel to facilitate adequate water absorption for rapid and uniform germination. All data for a certain lot of seed at a given temperature are based on the germination of 100 seeds.

The seedlings were grown in light-proof chambers at a relative humidity of approximately 85 per cent and at several temperatures with maximal fluctuations of ½° C. The relative humidity within the plugged test tubes was not measured, but it probably was equal to or slightly exceeded that of the chambers. At the end of 10 days the seedlings, except those grown at 18°, were removed to the laboratory, where they were grown for an additional four days at temperatures which ranged from 22° to 28° C. The removal to the laboratory was desirable because of the great elongation of the hypocotyls when the seedlings were grown in the dark for a longer period at these temperatures. This change in conditions could not have greatly influenced the results; for the critical period for seedling infection at these temperatures was during the first 8 days. The concurrent germination of the same lots in the laboratory also indicated that the laboratory temperatures

[1] Contribution of the Department of Botany and Bacteriology in Cooperation with the Division of Cotton and Other Fiber Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture. Technical Contribution No. 90, South Carolina Agricultural Experiment Station.

were favorable for infection. On account of the slow growth of the seedlings at 18°, it was necessary to grow them in the cases for two weeks, after which they were observed in the laboratory for an additional week. When the hypocotyls had attained a length of 70–80 mm. and the cotyledons had come in contact with the cotton plugs, the latter were removed. The first seedlings reached this length in approximately 12 days at 18°, 7 days at 22°, 4 days at 25°, and 3 days at 29°, 33°, and 36°. At the first four temperatures, the number of days indicates the approximate time required for complete emergence of the cotyledons from the testas. At 33° and 36°, many of the cotyledons did not emerge until after the plugs were removed, and the seedlings had been placed in the laboratory. These two temperatures, especially 36°, are too high for the optimal development of the cotyledons. When the agar at the higher temperatures became reduced in volume as the result of water loss, an attempt was made to maintain a water level at approximately that of the original agar gel by adding, on alternate days, the necessary amounts of the same salt solution as that used for making up the agar gel.

The four lots of seed of upland cotton (*Gossypium hirsutum* L.) used were of the 1938 crop. The varieties, the states in which they were produced, and the letters by which they and their seedlings will be referred to in this paper are as follows: A. Mexican Big Boll, North Carolina; B. Dixie-Triumph, South Carolina; C. Stoneville 2b, Mississippi; D. Stoneville 2b, Georgia. The germination of the first three lots under favorable conditions was approximately 90 per cent; that of the D lot was generally from 10 to 15 per cent lower. All four lots were externally infested by the anthracnose fungus (*Colletotrichum gossypii*) and *Fusarium moniliforme*. Lots A, B, and C had from 2 to 5 per cent of the viable seed infected internally by *C. gossypii*. There was apparently no similar internal infection of the D lot, for there was no infection of seedlings which developed from surface sterilized seeds.

The development of a primary root and a hypocotyl of sufficient length to raise the testa or cotyledons above the surface of the agar was the criterion for determining the percentages of germination. Those seeds which developed only radicles were counted as not germinating. Healthy seedlings were those which germinated normally and had no visible lesions on the cotyledons, hypocotyls, or radicles. At least 25 per cent of all lesions were examined microscopically to verify the macroscopic observations. However, all lesions atypical of those produced by the anthracnose fungus were examined microscopically to assure that other fungi were not overlooked. A seedling was recorded as dead when the cotyledons became withered and dry or the hypocotyl had been so injured by the fungus that it was no longer able to support the cotyledons in an upright position.

RESULTS

In the initial experiments the B and C lots were grown at 18°, 22°, 25°, 29°, 33°, and 36° C. to ascertain the temperature at which the anthracnose

fungus would cause the greatest injury to the seedlings. The total germination of both lots was about the same at the various temperatures and ranged from 81 to 91 per cent, except for an emergence of 75 per cent for the B lot at 18°. The extent to which the low germination of lot B at 18° may have been associated with a high infection of the non-germinating seed by *F. moniliforme* is uncertain. The number of living seedlings per 100 seeds after 14 days for both lots was in the order 33° > 36° > 29° > 22° > 25°. The number of healthy seedlings was in the same order, except for a slightly greater number of healthy seedlings at 36° than at 33°. Results for B lot are shown in figure 1. The number of seedlings infected but not killed at

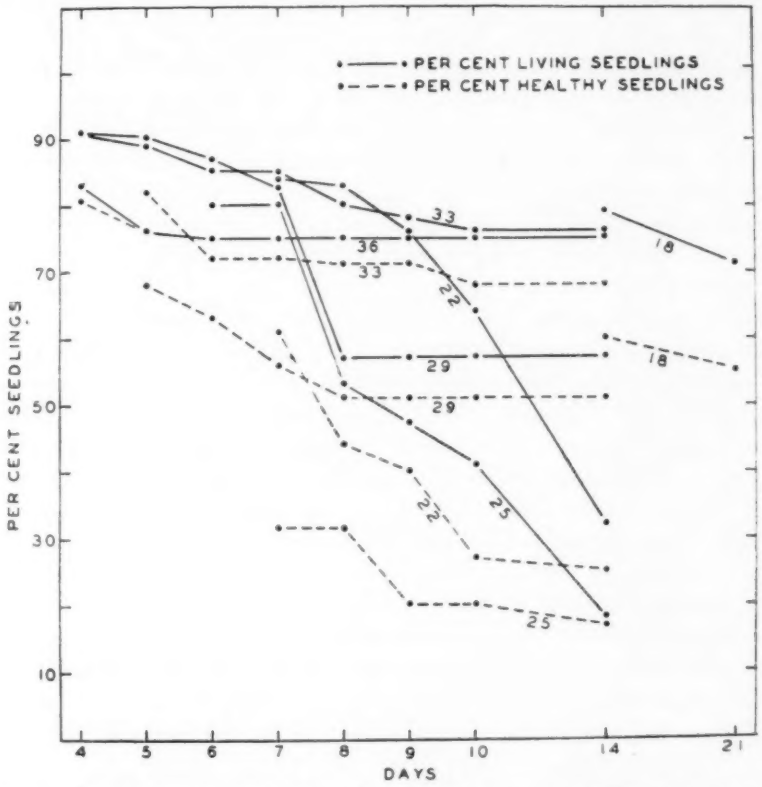

FIG. 1. The percentages of living and of healthy cotton seedlings of lot B (Dixie-Triumph) after incubation for the number of days indicated and at the temperatures denoted by the figures on the graphs.

each temperature is indicated by the difference between the solid line (percentage of living seedlings) and the dotted line (percentage of healthy seedlings). The first points to the left on the solid lines show the maximal percentages of germination; while the depression of these same lines to the right indicates the number of seedlings killed at successive intervals. The first points on the dotted lines were not recorded until the first visible lesions appeared on seedlings, so that healthy seedlings could be distinguished from infected ones.

Because of the much slower germination at 18° (a total of 85 per cent was attained on the 11th day) than at the higher temperatures, the results at the former temperature can be compared with the others only on the 14th day. At this time the percentage of living seedlings was largest at 18°, and the number of healthy seedlings for this temperature was midway between those at 29° and 33°. Relatively few of the seedlings which were alive after 14 days of incubation at 18° were infected and killed during the subsequent 7 days of incubation in the laboratory where the temperature was more favorable for infection. It is apparent that the reduction of the temperature from 22° to 18° decreases the susceptibility of cotton seedlings to injury by the anthracnose fungus, regardless of the slow growth of the seedlings at the lower temperature.

The number of seedlings infected at 33° and 36° (Fig. 2) was somewhat larger for the B lot than for the C lot during the first 7 days of incubation. After the 8th day the numbers for both lots at these temperatures were about the same. In contrast, at the lower temperatures the relative numbers of infected seedlings were approximately the same for the first day or two after the appearance of the lesions, but tended to differ after a longer period of incubation. Thus, on the 14th day the C lot had from 9 to 13 per cent more infected seedlings than the B lot at 22°, 25°, and 29°. There was a comparable difference between the lots at 18° C. on the 21st day.

A microscopic examination of the lesions showed that certain of the seedlings were infected by fungi other than *C. gossypii*. The anthracnose fungus did not infect the seedlings at 36° C., but 5 and 6 per cent of the cotyledons of the B and C lots, respectively, were completely rotted by a *Rhizopus* sp. within the first 7 days. The only other injury by *Rhizopus* was the destruction of approximately 4 per cent of the cotyledons of the seedlings of both lots at 33° and the destruction of one hypocotyl of the B lot at 18°. A *Penicillium* sp. was also found on 2 per cent of the severely injured cotyledons of the C lot at 36°. A *Rhizopus* sp., presumably the same as that infecting the cotyledons, was found on 25 to 50 per cent of non-germinating seeds of both lots at 18° and 36° and also on those of the B lot at 33°. The other temperatures apparently were not favorable for the development of this *Rhizopus* sp. or its growth was inhibited by the more rapid growth of other micro-organisms.

At 33° C. not more than 24 per cent of the seedlings of either lot were infected by the anthracnose fungus. The first lesions were visible on the 5th day, but few appeared after the 7th day; and all of the infected seedlings which died were killed before the end of the 10th day. About 40 per cent of the seedlings with lesions outgrew the injury. The seedlings killed by the fungus were generally those on which the lesions appeared earliest. In contrast, at the lower temperatures relatively few infected seedlings outgrew the lesions. At 29°, as at 33°, the first anthracnose lesions on the hypocotyls appeared on the 5th day. Most of these infected seedlings were killed before the 9th day, and only about 10 per cent of the seedlings infected at 29° outgrew the injury.

Although the anthracnose lesions appeared one to two days later at 22° and 25° than at 29° and 33° (Fig. 2), the greater number of seedlings were infected and killed at the two lower temperatures. At 22° and 25°, 80 and 87 per cent, respectively, of the seedlings of the C lot were infected; and 70 and 77 per cent, respectively, of the B-lot seedlings. Less than 10 per cent of infected seedlings were alive on the 14th day at both temperatures.

After it had been ascertained that the optimal temperature for the infection of the seedlings by the anthracnose fungus was approximately in the

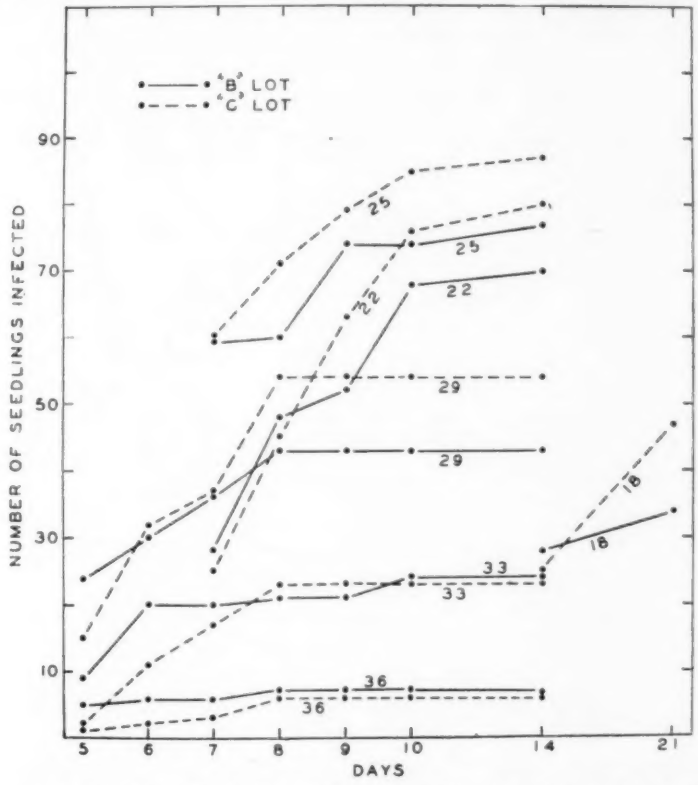

FIG. 2. The number of infected seedlings per 100 seeds of two lots of cotton seed ("B" and "C") after incubation for the number of days indicated and at the temperatures denoted by the figures on the graphs.

range of 22° to 25°, the four lots of seed were grown simultaneously at each of the two temperatures. At both temperatures the A lot gave the highest percentages of living and healthy seedlings (Fig. 3). At 25° the percentages of seedlings for the other 3 lots infected and killed were much the same, and the differences between them were small. At 22°, however, there were distinct differences among these 3 lots, the C lot producing the smallest number of living and healthy seedlings, the D lot the largest number, with the B lot intermediate. The proportion of healthy seedlings for the D lot was actually somewhat greater relative to the other two lots than is indicated on the graph on account of the 15 per cent lower germination of this lot.

The greatest difference in the incidence and severity of the lesions at 22° and 25° was shown by the A lot. At 22° after 14 days of incubation, there were 70 living and 63 healthy seedlings as compared to only 43 living and 38 healthy seedlings at 25°. Although the differences between these two temperatures for the other three lots were smaller, the graphs also show greater injury by the fungus at 25° than at 22°. This difference is further shown by the time required for the development of lesions. At 25°, there is a large increase in the number of infected seedlings during the 6th day with smaller increases for the next three days; while at 22°, there is a more

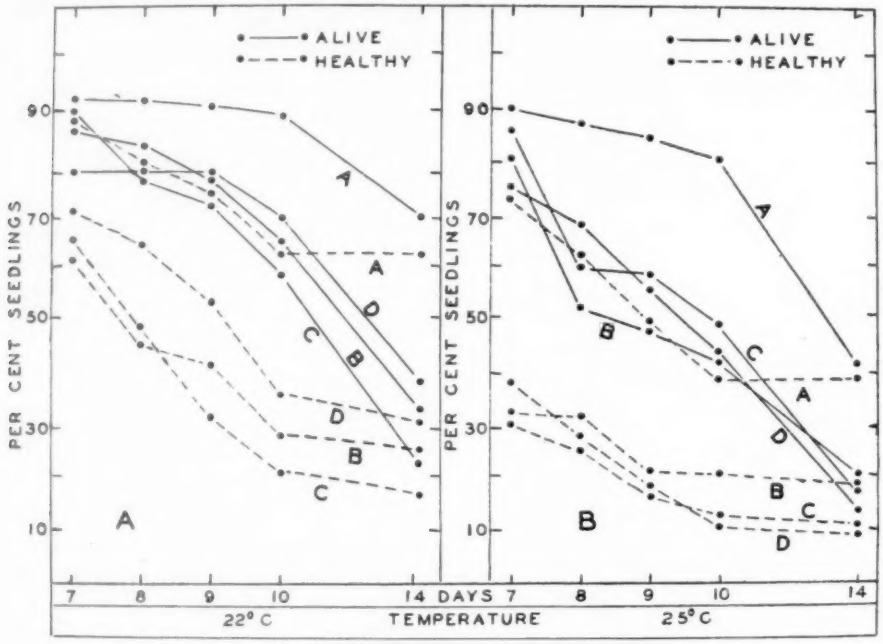

FIG. 3. The percentages of living and healthy seedlings of each of four lots of cotton seed after incubation for the number of days indicated at 22° C. (A) and at 25° C. (B).

gradual increase from the 7th to the 10th day. Much the same relative difference was shown in the time required to kill a given number of seedlings at these 2 temperatures.

Important differences were apparent among the four lots in the percentages of the seedlings which had anthracnose lesions on the cotyledons at the time when they emerged from the testas. The percentages for the A, B, C, and D lots at 25° were 40, 25, 25, and 0, respectively; while at 22°, they were 5, 10, 12, and 0, respectively. Most of the seedlings with lesions of this kind also had corresponding lesions on the hypocotyls. However, in the following instances about one-fourth of the seedlings with lesions on the cotyledons had no lesions on the hypocotyls: C lot at 22° and 25°, B lot at 22°, and A lot at 25°. The cotyledons of the A lot were especially susceptible to injury by the anthracnose fungus at 25° C.: 80 per cent of the infected cotyledons

were completely rotted before the 14th day. The percentages of cotyledons of the B and C lots rotted in this same period and at the same temperature were about one-half as great. The lesions which did not enlarge to involve the entire cotyledon mostly remained small and at the end of 14 days appeared as dry, brown spots 1–4 mm. in diameter. For the 3 lots in which anthracnose lesions appeared on the cotyledons, the lesions were generally larger as well as more numerous at 25° than at 22°. At the higher temperatures of 29° and 33°, less than 5 per cent of the cotyledons of the B and C lots were infected by the anthracnose fungus, and these lesions were invariably associated with corresponding lesions on the hypocotyls. At 18°, 5–6 per cent of the cotyledons of both lots were destroyed by *C. gossypii*, and 3 per cent by *F. moniliforme*. The destruction of cotyledons by a *Rhizopus* sp. and *Penicillium* sp. at 33° and 36° has been noted previously.

DISCUSSION

In 3 field plantings[2] in South Carolina in 1939 in which the same lots of seed were used, the surviving seedlings at 6 weeks after planting for the untreated seed of the A, C, D, and B lots were 53, 35, 28, and 25 per cent, respectively. The examination of the diseased seedlings showed that these differences were due largely to pre-emergence killing and damping-off caused by the anthracnose fungus. That the externally seed-borne anthracnose fungus was responsible for these differences in the number of surviving seedlings was also indicated by a comparison with a two-year-old lot of seed, "M," included in the field plantings. This lot was not infested by the anthracnose fungus and was of the same variety and strain as the A lot. Untreated seed of this two-year-old lot produced 68 per cent surviving seedlings, or 15 per cent more than A. The seed of the M, A, and C lots, after treatment with ethylmercuric phosphate, produced essentially the same percentage of seedlings, or 73, 73, and 67 per cent, respectively; while the percentages for the B and D lots were 60 and 46, respectively. Thus, treatment of the M, A, C, B, and D lots of seed increased the surviving seedlings by 7, 38, 91, 114, and 64 per cent, respectively.

The increases in seedlings in the field for the A and D lots approximated those which might have been expected from the percentages of their seedlings which were infected by the anthracnose fungus at 22° (Fig. 3). Similarly, the laboratory results would have indicated a greater response to seed treatment in the field for the C and B lots than for the A and D lots, but a greater response for B than for C would not have been expected. Thus, the germination of similar lots of seed at 22° by the laboratory method described should indicate their approximate response to seed treatment when planted in the field. Small differences among lots in the laboratory tests, however, may not be discernible in the field.

These results give no indication of the factors which may have been responsible for differences among the four lots. Ullstrup (6) has shown that

[2] These plantings were made in cooperation with the plant pathologists of 6 other states in a study of the effect of the characteristics of various seed lots on their response to seed treatment with ethylmercuric phosphate.

differences in pathogenicity exist among isolates of this fungus. Differences in spore loads, in degree of infection of the seed coat by the mycelia, and in innate resistance to infection by the cotton variety are not precluded.

The greater incidence of anthracnose lesions at 25° in these experiments than at 29° suggests that the optimal temperature range for seedling infection by *C. gossypii*, when naturally infested seeds are used, may be in the lower portion of the 25–30° range reported by Lehman (5) for inoculated seeds. His data on the growth of the fungus at various temperatures, however, coincide closely with the amount of infection at the several temperatures used in this experimentation; for temperatures of 25° to 30.5° were found by Lehman about equally favorable for the growth of *C. gossypii*, with the lower portion of this range the more favorable for conidial production. There was no growth of the fungus at 34.5°, and at 21° the growth was slower than at 25°. These temperature relations of the parasite seem to explain the absence of anthracnose infection of the seedlings at 36°, the low percentage of seedlings infected at 33°, the higher percentage infected at 29°, and the successively reduced infection at the two temperatures tested below 25° C.

Camp and Walker (3) and Lehman (5) supply comparable data for the germination and early growth of cotton seedlings. Their observations indicate that germination and seedling emergence is most rapid at 30° or slightly higher, is slightly less rapid at 25°, and is much slower at 20° C. A comparison of these data with the incidence of anthracnose lesions at the various temperatures indicates that the causal fungus has its maximal pathogenicity at a temperature, about 25°, which is near the lower limit of the temperature range for its maximal growth but is well below the optimum for the host.

The data by Walker (8) on the infection of onion by a closely related fungus, *Colletotrichum circinans*, indicate a different relation of temperature to disease production. The optimal temperature for disease production is 26–27°, while the fungus mycelium on agar grows best at 20–26°. This places the optimal temperature for disease production at the upper limit of the temperature range favorable for mycelial growth rather than in the lower portion of the range as for *C. gossypii*. The former parasite, however, is relatively more virulent at 31° than is the latter. This difference may be associated with the fact that temperatures above 30° are relatively more favorable for the rapid and normal growth of the cotton plant than they are for the onion.

Rhizoctonia solani, another fungus that causes damping-off of cotton seedlings (8), is most active at temperatures which are below the optimal for damping-off by the anthracnose fungus. The optimal soil temperature for the damping-off of cotton seedlings by *R. solani* is given as 17° to 23°, with little reduction in the number of seedlings killed until a temperature above 31.5° C. is reached. This *Rhizoctonia* also grows well over a wider range of temperature than does the anthracnose fungus. Field observations in South Carolina indicate that the rhizoctonia fungus is more active as a

parasite on cotton plants at temperatures below 20° than the anthracnose fungus; but that at higher temperatures their relative importance is reversed, especially when the planting of infested seed is followed by moderate temperatures associated with high soil moisture.

The rapid decay of a small percentage of the cotyledons by a *Rhizopus* sp. at 33° and 36° indicates that the causal fungus is most active at temperatures approximating those most favorable for *R. tritici* (9) rather than those for *R. nigricans*. No comparisons were made in culture of the various mycelia of the *Rhizopus* spp. found on the decayed cotyledons and on the nongerminating seeds at other temperatures.

SUMMARY

Cotton seed infested by *Colletotrichum gossypii* were germinated at 18°, 22°, 25°, 29°, 33°, and 36° C. Anthracnose lesions appeared earliest at 29° and 33°. Relatively small percentages of seedlings were infected and killed at 33°. At 29°, much larger percentages were infected and killed, but not so large as at 22° and 25°. The last temperature approximated the optimal temperature for maximal infection and injury to the hypocotyls and cotyledons. Reduction of the temperature to 18° greatly reduced the incidence of seedling infection. At 36°, seedlings were not infected by this fungus.

The temperature for the maximal damping-off of cotton seedlings by the anthracnose fungus was in the lower portion of the temperature range favorable for the growth of the fungus and below the optimal temperature range for seedling development. In these experiments, practically all seedlings infected at 25° and 22° C. were killed before the 14th day. At 33° many of the lesions remained small and did not greatly retard the growth of the seedlings. From 4 to 6 per cent of the cotyledons were destroyed by a *Rhizopus* sp. at 33° and 36°.

The differences among the four lots of seed as to the percentages of seedlings killed were somewhat greater at 22° than at 25° C. The percentages of the seedlings of the lots killed in the laboratory at 22° were indicative of the response of these lots to seed treatment in field plantings.

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TOBACCO DISEASE CONTROL BY CROP ROTATION[1]

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Rotation is one of the oldest methods of improving crop production, and agronomists have for many years carried on extensive field-plot studies to determine the rotation systems best adapted to particular crops and areas. It is recognized generally that rotation practices influence the incidence of plant disease, particularly soil-borne diseases; but it has not proven feasible to obtain much specific information on disease control from the usual rotation experiment, as Goss and Afanasiev (7) report. Pathologists have recommended rotation as a control measure for many diseases, but, lacking definite field data secured under controlled conditions, these recommendations have been based largely on studies of host range and field observations. It has been assumed that the reduction of diseases through crop rotations was essentially a problem of starving out disease-producing organisms by planting resistant or immune crops and by destroying susceptible weeds. The present writers used definitely laid-out field plots to evaluate rotation as a control measure for tobacco root knot (*Heterodera marioni* (Cornu) Goodey), bacterial or Granville wilt (*Bacterium solanacearum* E.F.S.), stem rot (*Sclerotium rolfsii* Sacc.), and Fusarium wilt (*Fusarium oxysporum* Schl. var. *nicotianae* Johnson). Work was initiated in 1926 in Georgia and has continued to date in that State, and also in North and South Carolina. All tests have been repeated at 2 or more locations. It is not the purpose of this paper to report in detail on these studies, or to recommend rotation practices for the specific diseases studied. Rather the writers propose to present some of the data to show that the problem of plant-disease control through crop rotation is extremely complex and that it is not safe to base recommendations on general assumptions.

The field areas used were first planted for one year to a susceptible crop to insure that infection was even and severe. The plots were of two types: (a) Inclosed plots, 12×24 feet, made of tightly joined creosote-treated boards that extended 8 inches below and the same above the ground surface. Units were separated by 4- to 6-foot borders of bare soil. (b) Field plots, of 1/40 to 1/5 acre in different series, laid out in the usual manner except that surface drainage was carefully controlled to prevent interplot washing. Precautions were taken in all cultural operations to avoid interplot movement of soil or crop remains. The inclosed plot series were set up primarily to eliminate all possible movement of disease between plots and hence to serve as a check on the larger field-plot series. The correspondence of disease

[1] Cooperative investigations of the Division of Tobacco Investigations, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture, the Georgia Coastal Plain Experiment Station, the North Carolina Department of Agriculture, and the Agricultural Experiment Stations of North and South Carolina.

counts secured in inclosed and field-plot series was very satisfactory throughout. Disease counts were recorded on the Disease Index basis, a value of 100 indicating a maximum of disease and 0 no disease. Plot layouts were of the standard randomized-block type.

ROOT KNOT

Root knot affects numerous cultivated crops and weeds. Because tobacco is very susceptible, it serves as an excellent indicator crop and so provides a measure of root-knot-nematode populations in the soil. Rotation has been a standard recommendation for root-knot control, and the use of crops such as Brabham cowpeas and velvet beans has been suggested.

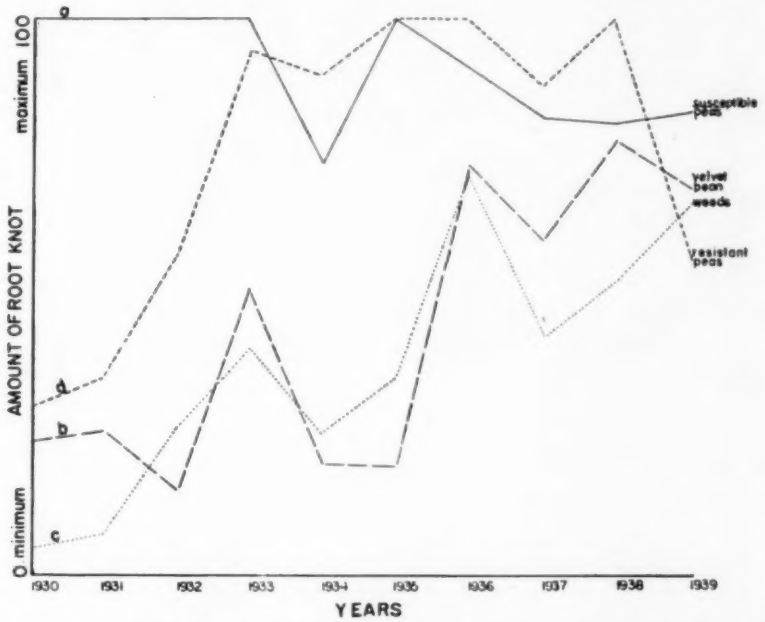

FIG. 1. Root knot on tobacco grown after 2 years of (a) susceptible Clay peas, (b) velvet beans, (c) native weeds, and (d) resistant Brabham peas. The results cover the period 1930 to 1939, inclusive, on land uniformly infested with root knot in 1926 at Tifton, Georgia.

Figure 1 shows the amount of root knot in flue-cured tobacco grown after 2 years of (a) susceptible Clay cowpeas, (d) resistant Brabham cowpeas, (b) velvet beans, and (c) native weeds over a 10-year period. During 1930 and 1931, tobacco after 2 years of resistant cowpeas (d), showed slight root knot, while tobacco after susceptible cowpeas (a) was severely affected. In 1933 this difference between susceptible and resistant peas disappeared, and throughout the remaining 7 years there was severe root knot on tobacco grown after both resistant and susceptible cowpeas. There was also a gradual increase in root knot on tobacco grown after velvet beans and after native weeds, with the result that the differences which were very striking

in 1930 were nonexistent or very slight in 1939. It might be assumed that resistant cowpeas, velvet beans, and weeds had become more and more susceptible to attack by root-knot nematodes, particularly in view of the recent important findings by Christie and Albin (2). Careful root examinations were made each year and there was no visible evidence that velvet beans and Brabham cowpeas were any more subject to root knot in 1939 than they had been in 1930. Both had slight traces of infection each year, but they otherwise were normal and healthy. Crops of susceptible cowpeas, however, were so severely affected by root knot that many of the plants died by midseason.

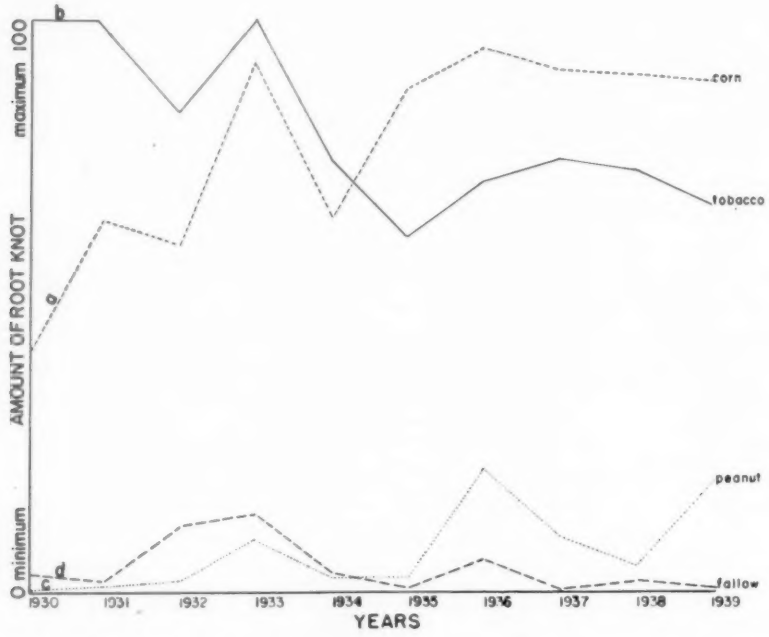

FIG. 2. Root knot on tobacco grown after 2 years of (a) corn, (b) tobacco, (c) peanuts, and (d) bare fallow, from 1930 to 1939, inclusive.

Corn also has been recommended for root-knot control (6), and under field conditions it is rare to observe galls on corn roots. Figure 2 shows the amount of root knot on tobacco grown after 2 years of corn, peanuts, and bare fallow, with a continuous tobacco check for comparison. Over the 10-year period corn was no better than continuous tobacco, and in repeated tests corn has never demonstrated any significant value in root-knot control. The disease actually decreased to some extent in continuous tobacco plots, while there was a definite increase in tobacco following resistant corn. The root-knot curves for tobacco after peanuts and bare fallow make a very sharp contrast with those for tobacco after the other crops. With bare fallow, starvation was highly effective in reducing root-knot-nematode popu-

lations, and the peanut crop was much more effective in starving out the nematodes than any of the others discussed.

In addition to the major crops grown during the summer, there are winter cover crops such as rye and oats that are highly resistant to nematodes, and others such as vetch and Austrian winter peas that are very susceptible. It would be expected that since the amount of root knot is reduced by bare fallow during the summer and generally increased by the culture of susceptible crops, the same principle would hold for winter practices. During

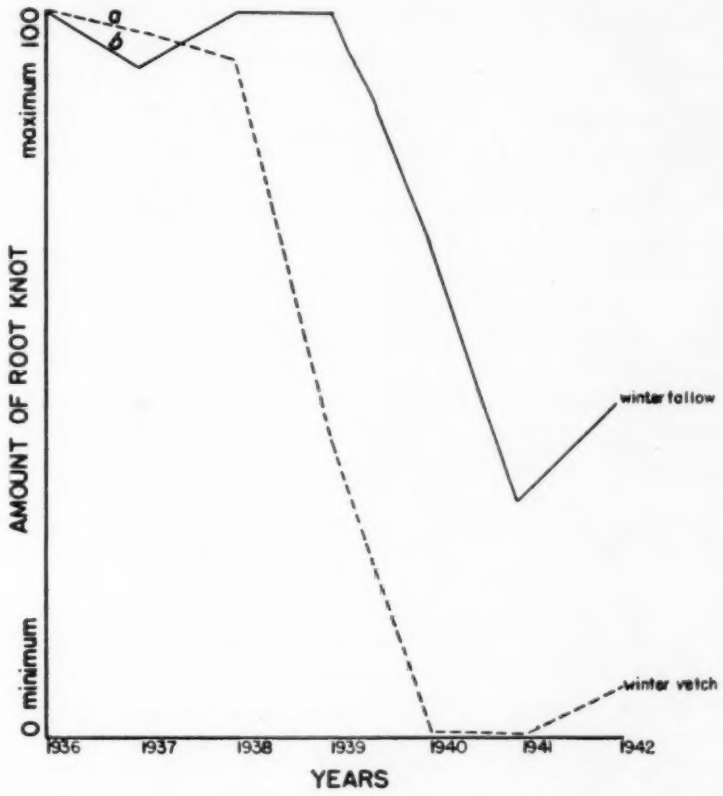

FIG. 3. Root knot in tobacco grown as a summer crop after (a) winter vetch and (b) winter fallow, from 1936 to 1942, inclusive.

the winter some plots were kept bare and some were seeded to winter vetch. The root knot in 7 successive summer crops of tobacco is shown in figure 3. In 1936–1938 root knot was very severe in all plots, but nematode population began to decline after the first 3 to 4 years. After 1938 there was much less root knot where tobacco was grown after a winter cover crop of vetch. During 1940–1942 tobacco grown after vetch was practically free of root knot, and the vetch, which had been severely galled during the early years, was also healthy. These findings are presented as indication that

culture of susceptible crops does not always increase soil nematode populations, and hence other very important factors are involved.

Additional basic problems associated with root-knot control by crop rotation can be illustrated with data from another experiment, at McCullers, N. C., laid out to provide maximum protection against interplot contamination. The units were 12×24 feet, and were tightly inclosed by board walls. Each treatment was replicated 4 times, and the mean values, with the appropriate error terms, are given in table 1.

TABLE 1.—*Influence of 2-year crop rotations on amount of root knot and yield of flue-cured tobacco, 1938 and 1940*

Tobacco grown after	Amount of disease[a]		Yield of tobacco (pounds green weight)	
	1938	1940	1938	1940
Crotalaria	38.8	42.8	43.4	70.5
Peanuts	41.0	37.3	31.0	46.8
Soy beans	84.8	59.0	40.4	58.4
Velvet beans	33.5	41.8	37.7	64.1
Sweet potato	100.0	95.8	34.6	40.4
Cotton	98.0	85.8	35.1	48.4
Corn	100.0	69.3	25.0	43.2
Weeds	84.5	83.3	31.3	47.4
Oats and weeds	86.3	86.5	33.7	45.6
Oats and fallow	35.3	35.5	30.8	43.3
Bare fallow	37.3	34.0	29.9	37.4
Tobacco	100.0	53.5	28.4	46.1
Difference required 5% level	8.76	17.45	5.14	4.31
for significance 1% level	11.77	23.44	6.90	5.79

[a] The amount of disease was recorded on the basis of 100 for maximum root knot and 25 for minimum root knot. (Each figure represents the mean value of 4 randomized replications.)

For all practical purposes a disease index value of 25 indicates complete freedom from root knot. The various crops were grown the first and third years and tobacco the second and fourth. One year of uniform tobacco cropping before the start aided in selecting an evenly and heavily infested area. The results in table 1 indicate that crotalaria, peanuts, velvet beans, oats plus bare fallow, and bare fallow were all very effective in reducing nematode infestation, and the differences in nematode control between these treatments were not significant. The weeds that followed oats were predominantly crab grass, and at this particular location small galls were observed on crab grass. It appears that crab grass following the oats nullified the effectiveness of oats, and the presence of crab grass in the weeds probably accounts for relatively poor results obtained with the weed rotation.

Practices giving good disease control and poor yields, however, would not be practical. Bare fallow has been suggested for nematode control and the data have shown it to be very effective; but in all experiments it has depressed yield. Tobacco grown after bare fallow was very free of root

knot but the yield usually was lower than that of more severely diseased tobacco following corn or even tobacco. Similarly, a healthy crop of tobacco was grown after oats followed by bare fallow, but the yield was less than with the severely diseased crop after oats and weeds. Bare fallow has reduced tobacco yield just as much as severe root knot, so that this treatment has not been a profitable one. Moreover, flue-cured tobacco quality was often lowered by bare fallow rotations, with the result that dollar returns per acre were still further reduced.

It is not unusual for fields with severe root-knot infection to produce excellent crops of tobacco, hence it is not safe to assume that a disease of this sort always reduces yield. Tobacco after sweet potatoes had severe root-knot infection in 1938 while tobacco after peanuts was practically free of root knot: the yield after sweet potatoes, however, was a little larger than after peanuts (Table 1). Root knot consequently is just one factor determining yield, and rotations giving maximum root-knot control would not necessarily be the most profitable for the grower to follow.

Assumption that failure to adopt the most stringent control measures will lead to constantly increasing damage does not appear to be justified in the case of a disease such as root knot. Thus, the experiment reported in table 1 was continued in 1942 and with an attack of only average severity there were no significant differences in the amount of tobacco root knot following the different rotations.

Despite the many complications, proper crop rotation remains by far the most practical and effective method of controlling root knot of tobacco. Control must be considered on a long-time basis, however, and the results from limited tests confined to a 3- or 4-year period can be quite misleading.

BACTERIAL WILT

Tobacco is very susceptible to bacterial wilt, and the list of host plants includes Irish potato, tomato, pepper, eggplant, peanut, and many weeds. Rotation has long been the main method of control recommended, but re-

TABLE 2.—*The occurrence of wilt-susceptible weeds in 1936 in relation to disease development in the subsequent tobacco crop in 1937*

Wilt-susceptible weeds per acre	Wilt in the tobacco crop	Wilt-susceptible weeds per acre	Wilt in the tobacco crop
Number	*Per cent*	*Number*	*Per cent*
0	27	6,758	28
40[a]	27	9,256	28
81	12	8,940[a]	19
162	36	74,792	3
324	36	116,278	44
728	27	140,024	11
761	5	389,976	35
1,011	35	574,312	35
1,492	10		

[a] Wilt-infected weeds were observed.

sults have been very uneven and failures to obtain control have frequently been attributed to growth of susceptible weeds in the immune crops. To test out this generally accepted theory weed counts were made in 17 separate wilt-infested fields in 1936 where growers expected to plant tobacco the following year. The results of the weed survey in 1936 and the percentage of wilt in the subsequent tobacco crops are given in table 2.

The range from 0 to 574,312 wilt-susceptible weeds per acre is very wide, but there was no correlation between the susceptible weed populations in

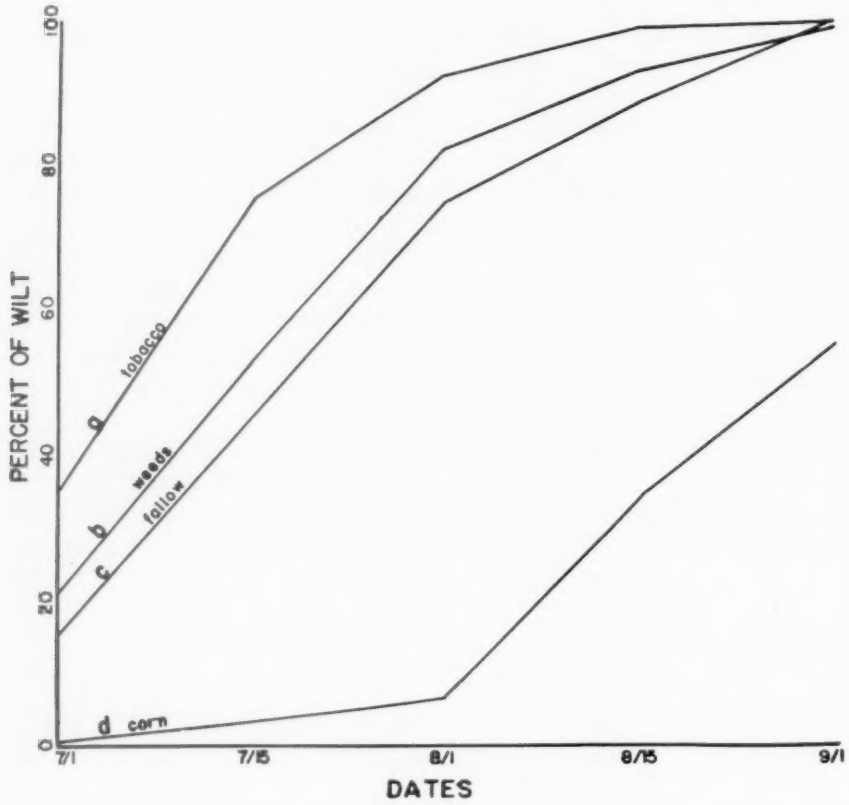

FIG. 4. The development of bacterial wilt in tobacco following (a) tobacco, (b) weeds, (c) bare fallow, and (d) corn, from July 1 through September 1.

1936 and the amount of wilt in the 1937 tobacco crop. Although many weeds are known to be wilt-susceptible, positively infected weeds are rarely observed under natural conditions. Wilt-infected weeds were observed in only 2 of the 17 fields in the survey. Further studies on the relation of weed host plants to the occurrence of wilt in tobacco were as follows: Plots were laid out in infested land and tobacco was grown after (a) corn and susceptible weeds, (b) weed-free corn, (c) crab grass and susceptible weeds, and (d) weed-free crab grass. Both corn and crab grass are immune to wilt.

Weeds in the crab grass grew poorly, but those in the corn grew vigorously and developed wilt following inoculation. These tests were repeated over a 4-year period and there was no difference in the amount of wilt developing in tobacco after weedy or weed-free preceding crops. It appears definitely established, therefore, that the failure of rotations with immune crops to give adequate wilt control in tobacco cannot be attributed to weed contami-

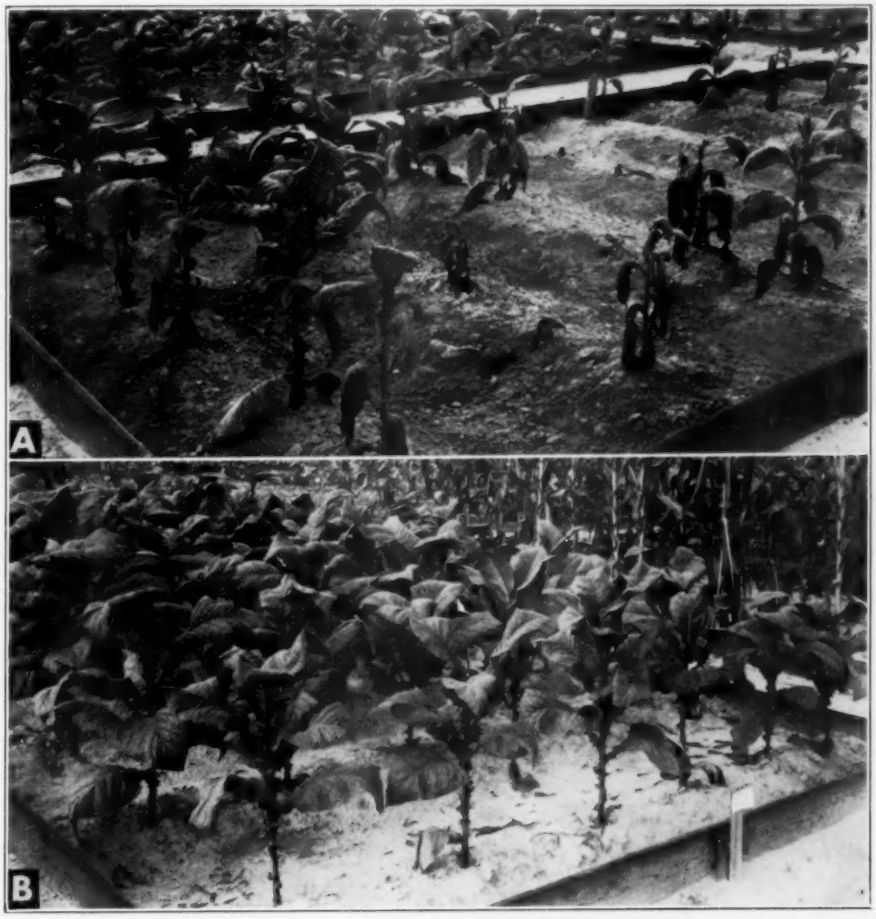

FIG. 5. A. Severe bacterial wilt in tobacco grown after bare fallow. B. The comparatively healthy tobacco produced after a year of corn.

nation. However, a natural weed fallow has been proven repeatedly to favor severe wilt in a succeeding crop of tobacco. The development of wilt in plots of tobacco grown after (a) tobacco, (b) natural weed fallow, (c) bare fallow, and (d) corn is illustrated in figure 4. The tobacco which followed a diseased crop of tobacco (a), was again severely diseased, as expected. However, tobacco after weeds (b) and bare fallow (c) was also severely affected. The general trend of these three curves is strikingly

similar, and on September 1 all these plots were 99 to 100 per cent affected. Corn greatly reduced the amount of wilt in the early summer. The heavy carry-over of wilt in the weed-fallow and bare-fallow plots cannot be explained on the basis of host plants, and the only possible explanation of the bare-fallow results is that this treatment provided conditions that favored persistence of the organism in the soil. Similarly the striking lack of infection after corn as compared with bare fallow (Fig. 4, c and d; Fig. 5) cannot be attributed solely to wilt immunity of corn.

STEM ROT

Root knot and wilt are caused by a nematode and bacterium, respectively, while stem rot is caused by the fungus *Sclerotium rolfsii* Sacc. Stem rot is widely distributed, but it is rare to observe a tobacco field with more than 5 per cent of the plants affected. Peanuts and soybeans, as well as tobacco, are attacked by stem rot, while crops such as corn and small grains are highly resistant. In the rotation plots at Tifton, Ga., tobacco-stem-rot counts have been made each year since 1926, when the plots were laid out, and similar records have been kept in other rotation series since established. The results have been consistent in that there was no relation between the character of the rotation and the amount of stem rot in tobacco. There was no more stem rot in tobacco grown after two years of stem-rot-susceptible peanuts than in tobacco grown after two years of resistant corn or after a complete bare fallow. At McCullers, N. C., where stem rot had been exceptionally severe on tobacco, in 1938, inclosed plots, 12×16 feet, were laid out in 1939 and rotations established with tobacco to follow tobacco, susceptible soybeans, and susceptible peanuts; also resistant corn and oats. All plots were heavily inoculated in 1939 and about 50 per cent of the tobacco, 15 per cent of the soybeans, and 70 per cent of the peanuts became infected. There was not a single case of stem rot in 1940 in the tobacco grown after these severely diseased crops, despite the fact that an abundance of sclerotia had been formed the previous year, an apparent confirmation of the statement by Wolf (15) that tobacco may remain free from stem rot even though the soil contains an abundance of the sclerotia. Although the 1940 tobacco had no stem rot, soybeans and peanuts grown after the 1939 diseased tobacco had, respectively, 40 and 32 per cent of stem rot in 1940. In 1941 there was again no stem rot in the tobacco plots, and the peanuts and soybeans had a trace to less than 5 per cent of disease. In 1942 the various continuous tobacco plots had up to a maximum of 14 per cent of stem rot, as compared with 0 to 8.7 per cent in the tobacco grown after other crops, but none of the differences were significant. The conclusion is that crop rotation had little effect on the amount of stem rot in tobacco, and that the use of such stem-rot-susceptible crops as peanuts and soybeans in tobacco rotations did not measurably increase the stem-rot hazard for later tobacco crops.

FUSARIUM WILT

Fusarium wilt is a comparatively new problem for tobacco growers of

the Southeast. At present it is important only in the Whiteville-Chadbourne area of North Carolina, but promises to become increasingly destructive unless growers take suitable precautions. Armstrong (1) recently showed that Burley tobacco is attacked by the same organism that causes Fusarium wilt of cotton. Smith and Shaw (13) found that certain physiologic races of the organism causing Fusarium wilt of sweet potatoes were able to attack both the green Orinoco varieties and the chlorotic Burley varieties of tobacco. The rotation experiments were not laid out with a view to studying the relation of crops to Fusarium-wilt occurrence but some very interesting observations were made. In four separate sets of rotation experiments, tobacco has been repeatedly grown after both sweet potatoes and cotton. In the Tifton, Ga., experiment, begun in 1926, Fusarium wilt occurred freely during the early years on both cotton and sweet potatoes. In 1937 it was first observed on tobacco, and in the sweet-potato rotation plots it has occurred in slightly increasing amounts each year since. Fusarium wilt has not yet occurred on the tobacco grown in the cotton rotation plots. At Florence, S. C., and other localities the rotation plots were initiated in 1936. Fusarium wilt has not been observed to date in the Florence experiment, but the disease was found on the Station farm where tobacco was being grown after a crop of sweet potatoes. At McCullers, N. C., Fusarium wilt was observed in tobacco the first year after sweet potatoes, and at Creedmoor, N. C., Fusarium wilt was observed in the 5th year of the sweet-potato-tobacco rotation. At none of these locations has Fusarium wilt been associated with cotton, or for that matter any crop other than sweet potatoes.

DISCUSSION

Of the four diseases studied, no two responded alike to crop rotations. There is considerable literature on root-knot control by rotation. Godfrey (6) concluded: "The most satisfactory method of combating the nematode in fields not planted to perennial crops is by the cultivation of immune crops for a period of two or three years and by carefully killing all weeds and susceptible plants in which the nematode can live. A desirable rotation is that where winter grains alternate with resistant cowpeas, Laredo soybeans, velvet beans, or beggarweed. Starving the nematodes by keeping the land free from all vegetation for two years is an effective control method, though often impractical." Le Roux and Stofberg (10), as the result of a 2-year test, concluded that nematode infestation could be effectively checked by eliminating all susceptible host plants. They also reported that bare fallow gave the best control and highest yields. Watson and Goff (14) found rotation the most practical method of control. They reported bare fallowing very effective, but discouraged the practice because of detrimental effects to the soil. In place of this, they recommended the use of root-knot-immune crops, particularly crotalaria and velvet beans. The writers have found root knot a good disease with which to study the comparative merits of various crop rotations. Previous workers have indicated preference for

crops thought to be completely immune to root knot, but the general practice has been to also recommend the use of crops regarded as moderately resistant, and no data have ever been given to show the comparative merits of the two groups. Figures 1 and 2 in the text bring out the fact that crops such as corn and resistant cowpeas, although they grew normally in heavily infested soils, did not starve out the nematodes, and actually were not superior to ordinary susceptible crops from the point of view of nematode control by crop rotation. Consequently, whether or not these resistant crops should be recommended for use on nematode-infested land depends on what the grower wishes to accomplish. If he desires to reduce the soil nematode population in preparation for growing tobacco or a susceptible truck crop, then his choice is limited to such apparently immune crops as peanuts, crotalaria, and oats. There were no significant differences between the reduction in nematode populations secured with these crops and the reduction secured with complete bare fallow. In all experiments, however, complete bare fallow reduced yields, and in one series bare fallow for merely a portion of the summer caused a greater yield reduction than the nematodes. Nematode control by bare fallow consequently would be neither practical nor profitable, particularly with light, sandy soils, where the nematode problem is most serious. So far as the writers are aware no one has called attention to the fact that under field conditions the nematode soil populations not only fluctuate from year to year but undergo gradual changes of great magnitude, even where the same cultural practices are followed many years. As would be expected, planting susceptible crops tended to build up maximum nematode populations, but with continued planting of susceptible crops the amount of root knot has not tended to remain constant. Maximum root-knot damage to plant roots in one series of continuous tobacco plots occurred during 1930 to 1933, and there was much less root knot in these same plots during 1935 to 1939. The corn-tobacco rotation plots in the same experiment showed an opposite trend, which would eliminate the possibility that these trends were associated with weather. Further evidence of the existence of important factors that influence root-knot-nematode soil populations and are entirely separate from crop susceptibility or resistance appeared in the experiments with tobacco following winter vetch or winter fallow. Despite the fact that vetch, as well as tobacco, is highly susceptible to root knot, the tobacco following vetch was practically free of root knot for the three years 1940 to 1942. Root knot decreased somewhat in the winter-fallow plots, but this tobacco continued to develop moderate to severe root knot. Linford, Yap, and Oliveira (11) have shown that it is possible to reduce nematode populations in the soil by the addition of organic matter, and their results and those of Drechsler (3) strongly suggest that nematode parasites may be an important factor in regulating nematode soil populations. The writers, however, have not obtained any marked nematode control by growing winter crops of rye, which under their conditions certainly added as much organic matter as the vetch. The note-

worthy difference was that in addition to organic matter the vetch built up a large reserve of nitrogen.

The results from the bacterial wilt rotation experiments were very different from those with root knot. Bare fallow, which was highly effective against root knot, was very ineffective against bacterial wilt. Tobacco planted after one year of complete bare fallow suffered almost as severely from wilt as tobacco grown immediately after a diseased crop of tobacco. On the other hand, the culture of corn for a year greatly reduced the amount of wilt in subsequent tobacco. Thus, immune-plant cropping and bare fallow, which gave equal and effective root-knot control, were not equally effective against bacterial wilt. The fact that many native weeds are susceptible to wilt has long been regarded as a complicating factor in controlling wilt by immune crop rotation. In actual tests, however, there was no correlation between the population of susceptible weeds and the development of wilt in subsequent tobacco. Furthermore, despite the fact that many weeds are known to be wilt-susceptible, it is rare to observe infection in natural weed growths on land known to be infested; and even when weeds were actually planted in crops of corn and permitted to become infected, the amount of wilt in the subsequent tobacco was not increased. It is assumed generally that allowing infested fields to "lay out" for several years fails to control wilt because some of the weeds are host plants. This assumption is open to question, and it may be that weed fallow merely favors survival of the organism in the soil. There is strong evidence that occurence and survival of *Bacterium solanacearum* in the soil depends to a large degree on factors other than culture of host plants. In the writers' experiments there was abundant infection remaining in soil after four years of complete bare fallow, and in general, immune crop rotations tended to greatly reduce but never eliminate the disease. Repeated culture of tobacco invariably increased wilt damage to tobacco, but Eddins (4) reports that in Florida, where the same disease affects potatoes, new fields are most severely affected and after several years of potato culture, wilt damage is greatly decreased. It would seem that control of bacterial wilt through crop rotation involves to only a limited degree the principle of "starving out" the organism. Certain crop rotation practices have been developed and used extensively in this country (5), and in the Dutch East Indies (9 and 12), which tend to greatly diminish the amount of wilt infection that carries over in the soil. A better understanding as to how such crops as corn tend to reduce wilt might make it possible to greatly improve our present rotation practices.

Despite the fact that *Sclerotium rolfsii* lives over in the soil from year to year, and that many common plants are susceptible to attack, such as peanuts and soybeans, while others, such as corn, are immune, extensive tests over a long period failed to show any connection between crop rotation and the occurrence of stem rot of tobacco. These negative results seem to indicate either that the organism is able to attack tobacco to only a limited de-

gree and under very special conditions, or that continuous culture of crops susceptible to *Sclerotium rolfsii* does not increase the amount of infestation in the soil and hence that parasitism is a minor factor in the life of this organism.

The results with Fusarium wilt seem especially to point to the great value of rotation plots set up for the study of disease control. Thus, recent work by Armstrong (1) and Smith and Shaw (13) clearly indicated the probability that the Fusaria causing wilt of either or both cotton or sweet potatoes might also attack tobacco. All 3 crops are extensively grown in the same areas of the Southeast. The rotation results support the conclusion that the culture of sweet potatoes and tobacco on the same land is hazardous and likely to lead to the establishment of a tobacco-Fusarium-wilt problem. Cotton can apparently be grown in the same rotation with tobacco of the Orinoco type without danger from Fusarium wilt.

CONCLUSIONS

Considering the general problem of disease control by crop rotation, these studies appear to justify the following conclusions:

The degree of disease control secured from crop rotation will vary considerably depending upon the year, the location, and the disease, as well as upon the nature and length of the rotation. A grower may rotate bacterial-wilt-infested land with the utmost care, and still suffer severe losses from wilt in occasional years, or in certain fields.

Resistant crops are not necessarily superior to susceptible crops as a means of reducing the carry-over of disease in the soil. Soil nematode populations were just as large after a crop of resistant corn as after a crop of susceptible tobacco. However, root knot was consistently reduced by the use of immune crops.

The occurrence of disease was generally related to the previous occurrence of host plants but the exceptions were important. There was as much stem rot in tobacco grown after stem-rot-immune corn as in tobacco grown after stem-rot-susceptible soybeans.

It was not true that complete elimination of all plants (bare fallow) favored most rapid elimination of all soil-borne parasites. Bacterial wilt infection survived better in the bare fallow plots than in plots which grew crop plants.

Rotations that affect the carry-over of soil-borne disease have other important effects on crop yields. Rotations favorable from the nutritional viewpoint may give high yields occasionally despite extensive disease development. Bare fallowing reduces root-knot-nematode populations but also depresses yields. Therefore, the figures for crop yield and the amount of disease are not always closely parallel, and rotations that are effective in disease control but reduce yields obviously have little value.

Rotation is the only practical method available for combating many serious diseases and the importance of developing rotation practices that

definitely aid in disease control is apparent. It is not possible to generalize with respect to rotation and disease control, for each disease presents its own special problem. The results from rotation experiments show wide fluctuations, hence data from isolated tests may be quite misleading. Reliable conclusions can be drawn only from controlled experiments under a variety of conditions and over a number of years. Considering the field of disease control as a whole, it appears that such data are conspicuously lacking.

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STEM RUST ON NEW WHEAT VARIETIES AND HYBRIDS[1]

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Since 1925 increasing numbers of stem-rust-resistant wheats have been bred and distributed in the United States. It has been customary to grow plant populations in the field under both natural and artificial rust epidemics for several years before a variety is considered adequately tested for rust reactions. In recent years there has been a growing tendency to test seedlings of the most promising materials to known physiologic races of rust maintained in pure culture in the greenhouse. Thus it is possible to determine the reactions of the new lines to the physiologic races of rust prevalent during the years in which the new hybrid populations were developing before the wheat is ever released. In spite of thorough testing, previously unimportant or new physiologic races or biotypes of races may be especially virulent on the new lines. Within a few years these virulent races may increase to such an extent that the new wheats no longer resist rust when grown in the localities for which they were bred.

An outstanding example of the change in rust population occasioned by the use of a new wheat variety is that of race 56 of *Puccinia graminis tritici* and Ceres wheat (6, 15, 19). Ceres was distributed in 1926, and by 1934 it was grown on an estimated four and one-half million acres (2). In 1928 race 56 was first identified, and by 1934 that race made up 35 per cent of the stem-rust population (19). Race 56 continued to occupy first place among the stem-rust races identified in succeeding years (19) and was especially virulent on Ceres. In 1935 and 1937 race 56 was so destructive to Ceres that farmers in the eastern part of the spring-wheat area reduced acreages, and the extensive plantings of that variety were pushed westward to the drier parts of the spring-wheat area where rust epidemics were less frequent. Another example of the interrelations of hosts and rust races is to be found in *Puccinia graminis avenae* and oats. New oat varieties, with stem rust resistance from a Richland parent, are susceptible to race 8 of oat stem rust. Stakman and Loegering (16) call attention to the greater prevalence of race 8 in 1943 and point out the probable increasing importance of that race if the new oat varieties supplant those now grown.

ARTIFICIAL STEM-RUST EPIDEMICS IN THE FIELD

Between 1939 and 1943 many of the stem-rust-resistant wheat varieties and hybrids were grown experimentally at University Farm, St. Paul,

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Minnesota, to see if they would remain rust-resistant when subjected to variations in environment and to early and heavy rust inoculum. Nearly 40 varieties of wheat considered highly or moderately resistant to stem rust under normal growing conditions and a few of the standard rust-susceptible varieties were obtained at various times through the courtesy of the Division of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture and the Division of Agronomy and Plant Genetics and the Division of Plant Pathology and Botany, Minnesota Agricultural Experiment Station. Pure cultures of 11 physiologic races of *Puccinia graminis tritici* were furnished by collaborators in the Division of Plant Pathology, Minnesota Agricultural Experiment Station and the Bureau of Entomology and Plant Quarantine, United States Department of Agriculture.

Two sowings, sometimes three, were made each year, the first one as early as weather and soil conditions permitted. Sowings were duplicated in 1941 and 1943 and quadruplicated in 1939, 1940, and 1942. Numerous borders of stem-rust-susceptible Hard Federation wheat surrounded each plot of randomized rust-resistant varieties; and the borders served to establish and increase the stem-rust inoculum put there at regular intervals by means of hypodermic syringes. Experimental varieties never were inoculated directly, but none of the test varieties were more than 25 feet from the first rust infection centers in the borders; and by the time the borders were severely rusted no experimental plant was more than 5 feet from rust inoculum.

Most of the physiologic races of stem rust chosen for study were prevalent throughout the United States between 1930 and 1940 (17, 18, 19): races 10, 11, 15, 17, 21, 34, 36, 38, 49, 56, and 147. Race 19 was used also, but since Hard Federation is resistant to this race, there was not sufficient inoculum of it to spread to the experimental varieties. Biotype B of race 15 has been used since 1940 because it has been isolated from a number of rust collections throughout the United States and is virulent on Rival, on Vernal emmer, and on some of the Kenya wheats (8, 19). Race 147 was included because of its virulence on Vernal emmer, used in durum-wheat-breeding work by reason of its resistance to many of the other physiologic races of stem rust.

Pure cultures of each race were grown in the greenhouse during March and April, so that sufficient quantities of viable urediospores could be collected and stored in an icebox for a few weeks prior to field inoculations. Between May 20 and 27, as soon as Hard Federation plants had 3 or 4 leaves, groups of 3 culms throughout the borders were inoculated by hypodermic syringe with freshly prepared suspensions of urediospores in distilled water. Each race was suspended and inoculated separately and the clumps were tagged, so that it was possible to ascertain that all races developed about equally well and contributed approximately equal quantities of inoculum for spread to the experimental varieties. Within 7 to 10 days after the first

border inoculations, rust spores were produced in abundance and the rust-resistant varieties were exposed to inoculum fully 8 to 12 days earlier than the natural and less abundant stem-rust inoculum usually is blown into the St. Paul area. Border inoculations, two or three times each week, continued through June 25, as long as any of the Hard Federation plants remained in the boot and suitable for retaining the suspension of inoculum. Rust spread rapidly in the susceptible borders and to certain of the experimental varieties. Most of the experimental varieties, however, did not rust for 12 to 18 days after rust inoculum was spreading from primary infection centers in the border; and the time elapsing depended to great extent on the temperatures prevailing in early June. Low temperature delayed sporulation.

All varieties were examined frequently and whenever large uredia, indicating susceptibility, appeared on a variety, collections were made to determine which physiologic races were attacking the variety and which were potentially dangerous for it. Small, poorly developed pustules, indicating a more or less resistant reaction, were disregarded because they were not considered dangerous or particularly injurious under ordinary conditions.

STEM RUST IN FOUR WIDELY GROWN WHEAT VARIETIES

In 1939 three varieties of hard red spring wheat were grown over extensive acreages in the United States (1). Approximately three and one-quarter million acres were planted to Marquis (C.I. 3641), more than three and one-half million acres to Ceres (C.I. 6900), and about five and one-half million acres to Thatcher (C.I. 10003). Rival (C.I. 11708) was relatively new in 1939 and was planted on about 1,000 acres (1, 21), but since that time has become more important and is more widely grown in the spring wheat area.

Marquis has been susceptible to many of the physiologic races of *Puccinia graminis tritici* described (20) and usually is heavily rusted in the field unless inoculum is scarce and environmental conditions are very unfavorable for stem-rust development. Ceres remained moderately resistant to stem rust for a number of years after it was released; but it is particularly susceptible to race 56, so that since 1934, when race 56 surpassed all other races in prevalence throughout the Mississippi Valley (18), Ceres often has been as heavily rusted as Marquis in the field. Thatcher has been resistant or moderately resistant to many of the stem rust races that attack Marquis; consequently it had largely replaced Marquis and Ceres by 1939. Between 1934 and 1940 rust infection was light in Thatcher unless that variety was planted unseasonably late. Rival also is resistant to many of the physiologic races of stem rust, and prior to 1940 it was not heavily rusted in the field.

Table 1 gives the amounts of stem rust in, and the stem rust races identified in collections from, the four varieties grown at University Farm, St. Paul, Minnesota, in plots provided with abundant inoculum each year from

TABLE 1.—*The development of stem rust and the physiologic races responsible for stem rust in Marquis, Ceres, Thatcher, and Rival wheats from 1939 to 1943 inclusive*

Variety and C.I. No.	Percentage[a] of stem rust in each sowing and physiologic races[b] of stem rust identified in collections															
	1939			1940			1941			1942				1943		
	Seeding date		Rust races	Seeding date		Rust races	Seeding date		Rust races	Seeding date			Rust races	Seeding date		Rust races
	Apr. 27	May 19		Apr. 19	May 17		Apr. 25	May 16		Apr. 14	May 4	May 25		Apr. 20	May 4	
Marquis, 3641	60–90	70–85	34 56	80–90	75–90	11^2 15^3 36^3 147^3 17^4 34^4 56^5	20–85	50–65	15 17 56	35–40	60–80	60–80	17 36^2 147^2 56^4 34^6 15^9	55	40–65	11 34 56 15^2
Ceres, 6900	40–70	50–80	34 56^3	70–95	75–85	11 17 36 49 15^2 147^2 56^6	40–65	35–55	10 21 34 36 15^3 17^3 56^4 147^4	12–35	30–60	50–80	34 147 17^3 34^4 56^4 15^5	15–55	15–40	15 147
Thatcher, 10003	5–12	10–30	15 34 36 38 56	15–45	30–65	11 34 56^2 15^{10}	Tr–8	Tr–5	10 36 56^3 147^4 15^5	8–10	20–40	55–60	36^2 15^{11}	.....	Tr–30	15^2
Rival, 11708	3–12	15–30	17 11^2 34^2	30–60	60–80	17 34 36 147 56^3 15^6	5–30	Tr–15	15 34 17^2 56^2 147^4	15–20	30–50	55–70	17 56 147 36^2 15^8	.....	15–45	15^2

[a] A range in percentage is given because there was considerable variation according to proximity to early inoculum.
[b] The exponent indicates the number of times a race was identified in collections from a variety.

1939 through 1943. Stem rust developed well on Marquis and Ceres every year, the percentages of infection varying with date of sowing, with proximity to early inoculum, and with environmental factors during a growing season. In 1940, 1942, and 1943, more stem rust developed on Thatcher and Rival under these conditions than had ever occurred before; and in 1940 and 1942 there was definitely more rust in late plantings of these two varieties than in early plantings. In 1940 there was 65 per cent stem rust on late-sown Thatcher and 80 per cent on Rival; and in 1942 there was only slightly less on late sowings of both varieties.

All four varieties were infected with several different physiologic races of stem rust, from 2 to 7 races being identified each year in collections from

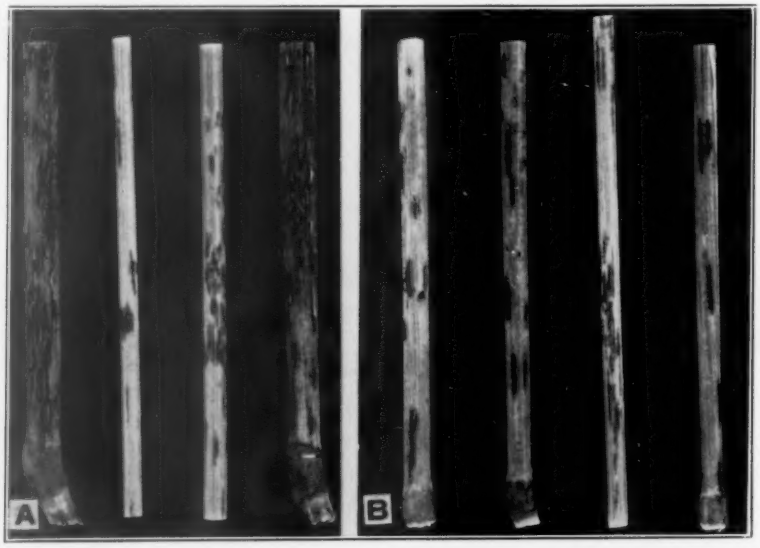

FIG. 1. The susceptible reactions of leaf sheaths and peduncles of (A) Rival and (B) Thatcher wheats to race 15 B of *Puccinia graminis tritici*. From field plots at University Farm, 1943.

a single variety. As reported previously (6, 15, 19), Ceres was an exceptionally good host for race 56, and in most years that race predominated in the rust collections from this variety. On Thatcher and Rival, however, race 15 predominated between 1940 and 1943; and in 1943 it was the only race obtained from these two hosts. Race 15 has a relatively wide host range, for it attacks 11 of the 12 varieties used as differential hosts for physiologic races of *Puccinia graminis tritici*, only Khapli emmer being resistant to it (20). Rival wheat acts as a differential host for biotypes of race 15, because it is resistant to biotype A and susceptible to biotype B (8). Biotype B of race 15 was first included in the inoculum in 1940, and in that year and subsequently it was a definite factor in causing rust on these four wheat varieties and many others in the experimental plots. Susceptible reactions occurred in Thatcher and Rival (Fig. 1) as well as in Marquis and Ceres,

all uredia on leaf sheaths and peduncles being well developed and without the signs of resistance that are so evident when Thatcher and Rival are attacked by less virulent rust races.

STEM RUST IN VARIETIES OF COMMON WHEAT CONSIDERED RESISTANT TO STEM RUST

A number of the new hybrid varieties that have been resistant to stem rust under farm field conditions were available in 1940 or 1941, among which were the ten varieties or selections listed in table 2. Of these, Hope probably has been the most resistant to stem rust over a period of years and while

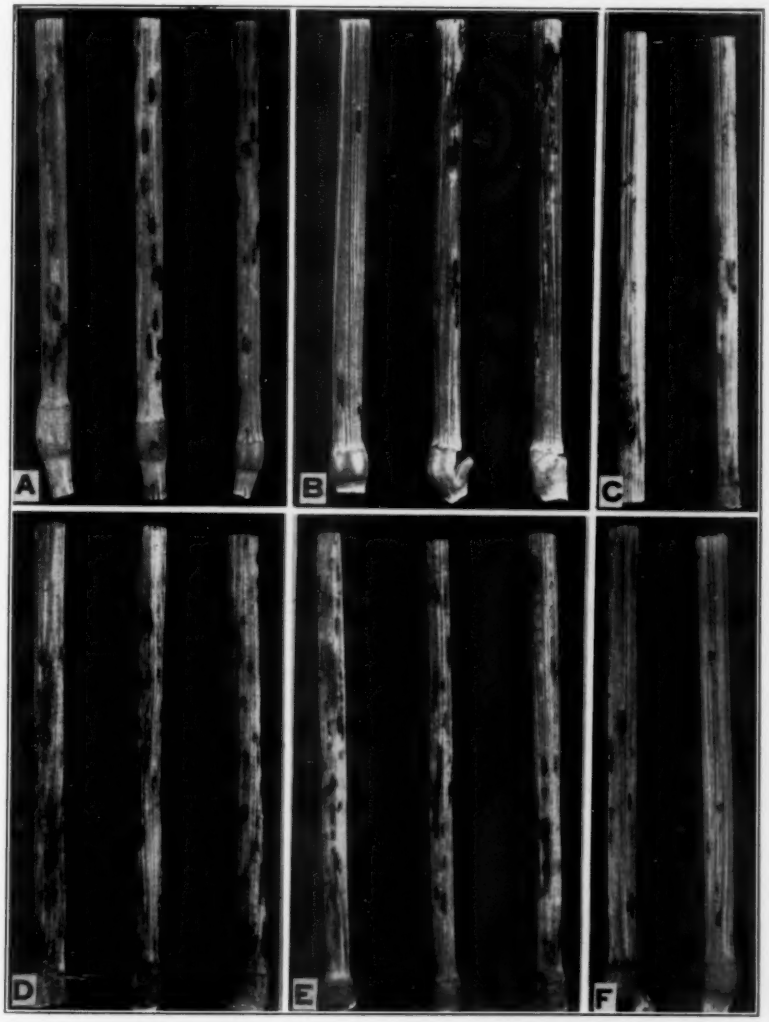

FIG. 2. Susceptibility to stem rust in (A) Apex, (B) McMurachy's Selection, (C) Renown Selection, (D) Premier, (E) Pilot 13, and (F) Regent Selection at University Farm, 1943.

TABLE 2.—*The development of stem rust and the physiologic races responsible for stem rust in varieties of common wheat usually considered resistant to stem rust*

Variety and C.I. No.	Percentage[a] of stem rust in each sowing and physiologic races[b] of stem rust identified											
	1940		1941			1942				1943		
	Seeding date	Rust races	Seeding date		Rust races	Seeding date			Rust races	Seeding date		Rust races
	Apr. 19		Apr. 25	May 16		Apr. 14	May 4	May 25		Apr. 20	May 4	
Apex, 11636	20–30	.....	10–30	2–15	147	10–20	30–40	50–60	15^2	15–20	18–30	15^2
Hope, 8178	Tr–20	.....	Tr	Tr	34	3–8	5–15	10–35	36^2 15^3 34^3	Tr–15	5–20	56 15^2 34^2
McMurachy's Sel., 11876	.....	.....	Tr–18	Tr–10	15 21 11^2 147^2	0–15	15–18	15–35	34 15^3	12–15	3–15	56 34^2 15^1
Mercury, 11872	15–30	.....	18–20	2–20	17	10–35	35–45	55–70	34 15^1	20–25	20–25	34 15^2
Merit 3, 12036	.....	.....	1–8	Tr–2	.....	Tr–3	15–30	50–60	15^3	5–15	5–15	15^2
Premier, 11940	5–20	15 56	2–12	Tr	17	8–15	35–40	20–60	15^3	3–30	5–18	15 34^1
Pilot 13, 11945	.....	.....	5–20	Tr–10	11 21	3–12	18–35	50–60	15^3	20–25	20–25	34 56 147 15^1
Renown Sel., 11947	15–25	15	10–25	Tr	.....	Tr–3	10–25	35–50	15^3	5–20	8–15	34 15^2
Regent Sel., 12070	2–25	.....	2–3	Tr	.....	Tr–2	5–25	15–35	56 15^2	Tr–10	Tr	34 15^2
Vesta, 11712	10–25	34	30–40	Tr–8	34	12–20	25–35	55–65	15^3	10–20	10–20	11 15

[a] A range in percentage is given because there was considerable variation according to proximity to early inoculum.
[b] The exponent indicates the number of times a race was identified in collections from a variety.

it has too many defects to be an acceptable commercial wheat it has been used extensively as rust-resistant parental material for breeding new wheats. McMurachy's Selection also has been considered an excellent source of stem-rust resistance (10, 13). Most of the others listed in table 2 were bred from crosses made between 1925 and 1935, and in some cases there has been re-selection for greater stem-rust resistance. All are considered moderately resistant to stem rust at present, but not all have been released or are recommended for commercial production. Apex, Renown, and Regent are grown commercially in Manitoba, Saskatchewan, and Alberta in Canada (10) and to a limited extent in the United States. Pilot has been grown on small acreages in the western part of the spring-wheat area of the United States (1, 21), and Vesta has been recommended for the western part of North Dakota (22).

The highest percentages of stem rust developed on varieties of this group in the late plantings of 1942. In that year even Hope, McMurachy's Selection, and Regent Selection had moderate rust infection and the other varieties were heavily rusted. Race 15 biotype B was responsible for most of the infection in 1942 and 1943, although race 34 was about as virulent as race 15 on Hope. In 1943 race 34 was the second most prevalent race according to identifications of rust from these varieties; and on Hope and Premier it was identified more often than race 15.

There were susceptible reactions in most of these varieties, for uredia were large and sporulated well and there was no chlorosis of tissues around the uredia (Fig. 2). Renown and Regent Selections were only moderately susceptible in the field, because many of the uredia were rather small although sporulation was good and there was no chlorosis (Fig. 2). Since race 15 B was identified in collections from all these varieties in the field plots, seedling reactions were determined in the greenhouse. No variety in this group was resistant to race 15 B: the infection type on seedlings of Premier and McMurachy's Selection was 3−, but it was 3++ to 4+ on other varieties.

STEM RUST ON RECENT HYBRIDS AMONG THE COMMON WHEATS

Multiple crossings have been made to combine desirable characters for quality and disease resistance of several promising wheats in one or more hybrids that will be satisfactory in all respects. Newthatch (C.I. 12328), which is now being released to farmers in Minnesota, was produced by crossing Hope and Thatcher and backcrossing the progeny to Thatcher twice. Several lines of similar hybrids exist, with varying degrees of resistance to both stem and leaf rusts. In other instances Thatcher or Double Cross have been crossed with H 44, the sister selection of Hope, or with Merit which originated from an H 44 × Ceres cross. Mida (C.I. 12008) was produced by crossing a selection from a Ceres-Hope-Florence combination with a selection from a Ceres-Double Cross combination. And in another case Reliance, Hope, and Comet have been used to produce a hybrid (C.I. 12050).

TABLE 3.—*The development of stem rust and the physiologic races responsible for stem rust in several complex hybrids among the common wheats*

Variety or hybrid and accession No.	Percentage[a] of stem rust in each sowing and physiologic races[b] of stem rust identified in collections from varieties									
	1941			1942				1943		
	Seeding date		Rust races	Seeding date			Rust races	Seeding date		Rust races
	Apr. 25	May 16		Apr. 14	May 4	May 25		Apr. 20	May 4	
Mida, C.I. 12008	.....	.....	.....	10–25	40–50	60–70	34 56 15[5]	25–40	20–35	15[5]
Reliance-Hope × Comet-Reliance-Hope, C.I. 12050	.....	.....	.....	18–25	40–55	65–70	.....	30	20–40	34 15[5]
Newthatch, C.I. 12328	.....	.....	.....	.....	.....	.....	.....	20–25	10–18	34 36 15[5]
Hope × Thatcher, C.I. 12199	2–3	Tr–5	.....	3–15	30–45	55–80	15[3]	5–25	8–15	34 15
Hope × Thatcher, C.I. 12043	5–15	Tr	15	1–10	18–35	30–45	15[4]	8–25	15–18	15
Hope × Thatcher, C.I. 12251	2–30	Tr–5	15	5–20	40	45–50	34 15[3]	10–25	3–30	15
Hope × Thatcher, C.I. 12044	Tr–2	Tr–2	.....	Tr–5	15–35	30–40	15[4]	10–20	10	34 15[2]
D. C. × H44, II–29–52, Minn. 2681	Tr–3	Tr–6	.....	3–12	25–30	30–60	15[2]	.....	.....	.....
D. C. × H44, II–28–27, Minn. 2682	5–40	Tr	15	Tr–8	.....	45–75	15	.....	.....	.....
Merit × Thatcher, Minn. 2705	.....	.....	.....	5–18	35–45	45–55	15[3]	15–25	8–15	15[2]

[a] A range in percentage is given because there was considerable variation according to proximity to early inoculum.
[b] The exponent indicates the number of times a race was identified in collections from a variety.

Many of these hybrids were supplied the author[2] and the results with a few of them are given in table 3. Most striking is the virulence of biotype B of race 15 and the preponderance of that race in all rust collections from

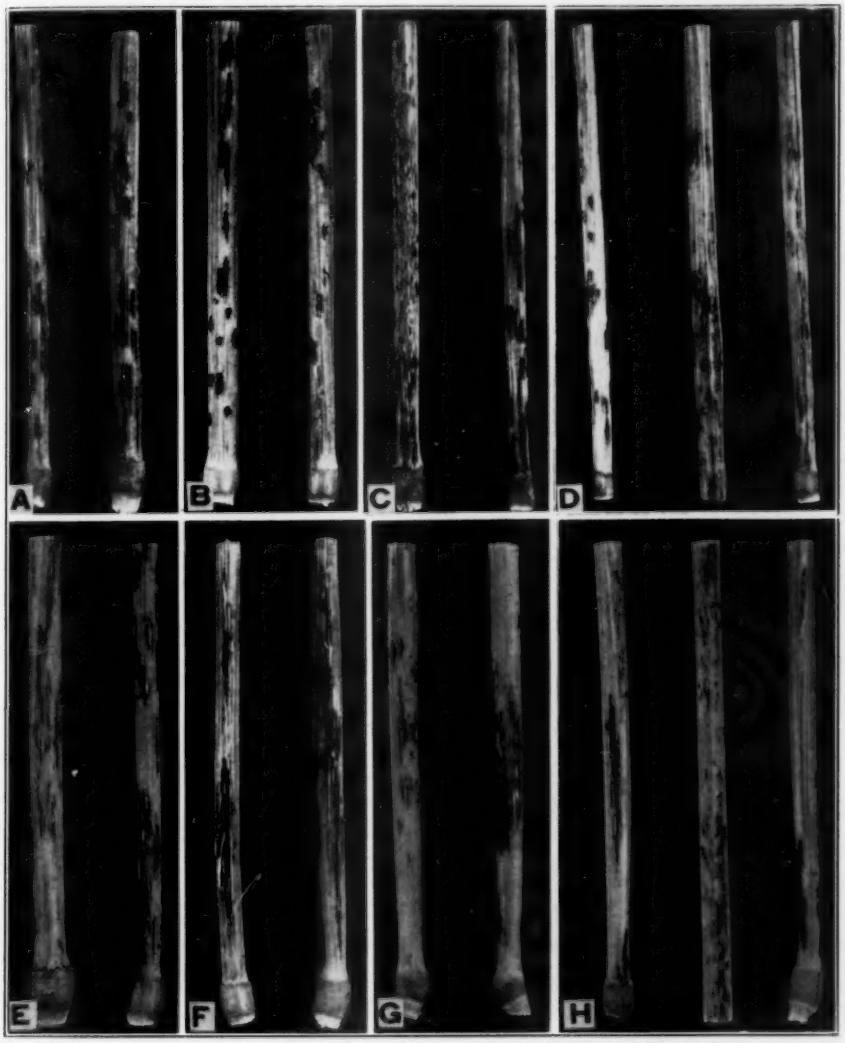

FIG. 3. Susceptibility to stem rust in (A) Merit × Thatcher hybrid (Minn. 2705); (B) a Reliance-Hope × Comet-Reliance-Hope hybrid (Minn. 2716 or C.I. 12050); (C) Mida; (D) Newthatch; (E) Iumillo; (F) Stewart; (G) a Mindum × Vernal hybrid (Minn. 2717); and (H) *Triticum timopheevi* at University Farm, 1943.

these hybrids from 1941 through 1943. The susceptible reactions of four of the hybrids from the field plots are shown in figure 3.

[2] Dr. E. A. Ausemus, Division of Cereal Crops and Diseases, United States Department of Agriculture, kindly furnished seed of these hybrids.

STEM RUST ON DURUM WHEATS, ON DURUM-EMMER HYBRIDS, AND ON TRITICUM TIMOPHEEVI

The durums Acme and Iumillo have considerable resistance to stem rust. Iumillo was crossed with the common wheat Marquis to obtain the stem-rust-resistant Marquillo (5); and Iumillo also went into the double cross (Marquis-Kanred × Marquis-Iumillo) from which Thatcher came (4). In years past both Acme and Iumillo have been lightly rusted in the field, but in 1942 both varieties were heavily rusted, especially in late plantings, and race 15 B was identified in all rust collections from the two wheats (Table 4). In 1943 the percentages of rust infection were not so high, but race 15 B again seemed to be responsible for a large part of the rust.

TABLE 4.—*The development of stem rust and the physiologic races responsible for stem rust in durum wheats, in Vernal emmer, and in Triticum timopheevi*

Variety or hybrid and C.I. No.	Percentage[a] of stem rust in each sowing and physiologic races[b] of rust identified						
	1942				1943		
	Seeding date			Rust races	Seeding date		Rust races
	Apr. 14	May 4	May 25		Apr. 20	May 4	
Acme, 5284	10–40	40–50	60–80	15^3	20–25	15–18	15^2
Iumillo, 1736	8–30	15–40	65–80	15^5	5–25	15–25	11 15^2 34^2
Mindum, 5296	10–30	55	65–75	.....	30–40	25–35	15 34^2
Vernal, 3686	Tr–20	30–40	35–55	.....	2–8	15–20	15^2
Carleton, 12064	15–30	60–70	60–70	15^5	25–30	20–30	15^3
Stewart, 12066	10–35	55–65	55–70	34 15^7	25–30	20	15^2
Mindum × Vernal, 12225	8–30	40–50	40–80	15^3	15–20	10–25	147 15^2
T. timopheevi, 11802	Tr–15	3–25	25–40	15^7	10–15	10–25	15^4

[a] A range in percentage is given because there was considerable variation according to proximity to early inoculum.

[b] The exponent indicates the number of times a race was identified in collections.

Rust-resistant durum wheats have been bred for the Red River Valley area of Minnesota and North Dakota and other sections of North and South Dakota by crossing the high-quality Mindum variety with the stem-rust-resistant Vernal emmer, then backcrossing the progeny to Mindum. Two new varieties, Carleton, C.I. 12064, and Stewart, C.I. 12066, have been named and released by the North Dakota Agricultural Experiment Station in cooperation with the Division of Cereal Crops and Diseases in the United States Department of Agriculture (14). They resemble the Mindum parent in growth habit and are late in maturing at St. Paul. In 1942 the parents and hybrids were heavily rusted in the author's experimental plots and biotype B of race 15 was prevalent (Table 4). Again in 1943, when stem rust was moderately heavy, race 15 B was more often isolated than any other

race. Both parents are susceptible to both known biotypes of race 15, so one would not expect the hybrids to be resistant. Whether biotype B may be more destructive to these wheats than biotype A over a period of years is not yet known, but all the hybrids had very susceptible reactions to stem rust in 1943 (Fig. 3), when race 15 B was prevalent in the experimental field plots.

Triticum timopheevi, considered highly resistant to stem and leaf rust and to several other diseases as well, was brought to the United States for

TABLE 5.—*The development of stem rust and the physiologic races responsible for stem rust in six Kenya wheats in 1943*

Kenya hybrid and accession No.[a]	Percentage[b] of stem rust in each sowing and physiologic races[c] of rust identified		
	1943		
	Seeding date		Rust races
	Apr. 20	May 4	
Kenya 122DI(I)(L), R.L. 1373, Minn. 2693	2–15	Tr–2	15^2 34^3
Kenya 117EB(I), R.L. 1374, Minn. 2694	15–30	15–18	15 34
Kenya 117B5B(E), R.L. 1375, Minn. 2695	15–20	10–25	56 15^2 34^2
Kenya 117K16A(L), R.L. 1376, Minn. 2697	25–35	10–35	56 15^2 34^2
Kenya 117I.5F.(L), R.L. 1377, Minn. 2696	8–30	15–25	34 15^2
Kenya 58	15–25	Tr–1	15 34^4

[a] The first designation for each wheat is that used by G. J. L. Burton of Kenya Colony, East Africa; the second is the number used by the Dominion Rust Laboratory, Winnipeg, Manitoba, Canada; and the third is the number used by the Minnesota Agricultural Experiment Station. Kenya 58 was obtained from M. N. Levine of the Division of Cereal Crops and Diseases, United States Department of Agriculture, who received it from R. J. Lathbury of Kenya Colony.

[b] A range in percentage is given because there was considerable variation in rust according to proximity to early inoculum.

[c] The exponent indicates the number of times a race was identified in collections from a variety.

breeding purposes, so that it might be crossed with other species of *Triticum* and contribute genes for its high degree of disease resistance to the progeny. The development of stem rust on *T. timopheevi* in 1942 and its moderate susceptibility to race 15 B has been reported by the author (3). In 1943 *T. timopheevi* was again rusted (Fig. 3), although not so heavily as in 1942; and race 15 B was the only race identified in four collections (Table 4).

Both Iumillo and *Triticum timopheevi* probably may be regarded as differential hosts for the two biotypes of race 15. In various experiments in Canada one or both of these wheats have been very resistant to race 15 (7,

11, 12), and it has been assumed that the Canadian race 15 was different from the race 15 B used in the present experiments. Seedlings of Iumillo inoculated in the greenhouse at St. Paul, Minnesota, had a 3++ infection type with race 15 B, while those of *T. timopheevi* had a 3+ infection type.

STEM RUST ON KENYA WHEATS IN 1943

Some of the wheats from Kenya Colony in East Africa have been reported very resistant to stem rust in North America (13) and in Australia (9). They are considered good breeding material in this respect. Six of the Kenya wheats were planted in the experimental plots in 1943. Nearly all were late maturing and were susceptible to leaf rust. Stem rust infection was not particularly heavy (Table 5), but some of the stem rust uredia

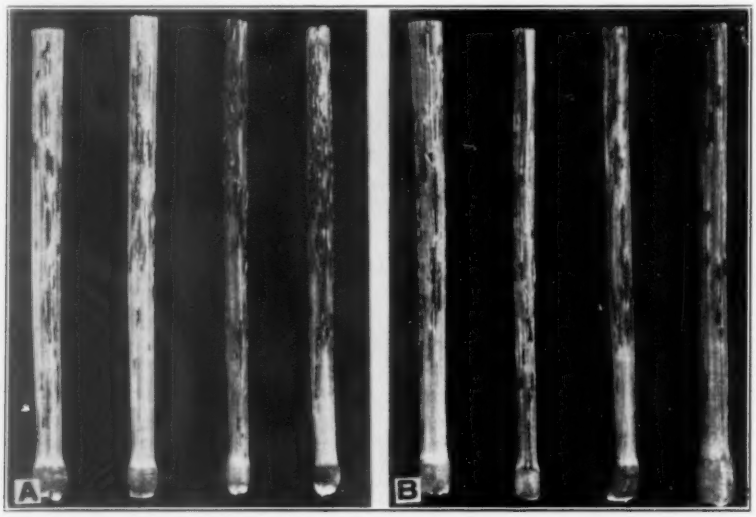

FIG. 4. Susceptibility to stem rust in (A) Kenya 117EB(I) and (B) Kenya 117K16A(L) at University Farm, 1943.

were large and sporulated so abundantly as to indicate a definite susceptibility (Fig. 4). Races 15 B and 34 were identified from rust collections from these wheats, and race 34 predominated. When race 15 B was identified, it usually was mixed with race 34 and constituted the smaller proportion of each collection. Peterson, Johnson, and Newton (13) found the Kenyas resistant to races 15 and 34 at ordinary temperatures and Watson, in genetical studies on the inheritance of the Kenya type of stem rust resistance, reported (23) his parental Kenya resistant to races 15 and 34. Later, however, Johnson and Newton (7) found that high temperatures made a Kenya selection, as well as some other wheats, susceptible to races 15, 29, and 56. They believe that temperature differences are responsible for many of the discrepancies in rust reactions of such wheats as the Kenyas at Winnipeg, Manitoba, at St. Paul, Minnesota, and at Manhattan, Kansas. Kenya seedlings were inoculated by the author with the biotypes of races 15 and 34

available at University Farm. Five of the Kenya hybrids were moderately susceptible to race 15 B, with 3 or 3+ infection types; while the hybrid Kenya 58 had 0; and 1 infection types and was resistant to race 15 B. When inoculated with race 34, seedlings of three of the Kenyas (Minn. 2695, Minn. 2696, and Minn. 2697) were moderately susceptible, with 3 or 3+ infection types; but seedlings of one Kenya (Minn. 2693) were resistant, with 1+ infection type. In one hybrid (Minn. 2694) some of the seedlings had 1++ infection types with race 34, while other seedlings in the same pots had 3+ infection types.

THE REACTIONS OF WINTER WHEAT HYBRIDS TO RACE 15 B

For the winter-wheat areas of the United States certain stem-rust-resistant hybrids have been produced by crossing a resistant parent such as Hope or Marquillo with winter wheats such as Oro, Tenmarq, Kawvale, or a Turkey selection. Seventeen such hybrids and 4 Kawvale × Tenmarq hybrids, together with the variety Pawnee (C.I. 11669), were sent to Minnesota for resistance studies.[3]

In 1943 vernalized seed of the 22 winter wheats were planted adjacent to the experimental plots of spring wheats. Thus the winter wheats were exposed to the rust inoculum provided for the other plots, and all 22 were well rusted by the time the plants had stooled. Since vernalization did not result in a general production of fruiting culms, stem rust on the leaf blades was collected for the determination of races attacking the hybrids. Race 15 B was identified in 14 of the 21 collections and race 34 occurred in 8 of the 21. Field reactions agreed very well with the greenhouse reactions of seedlings to these rust races. Seedlings of all were very susceptible to race 15 B, with infection type varying from 3+ to 4+; and all were moderately susceptible to race 34, infection types in that case varying from 3= to 4–.

DISCUSSION

Epidemics of wheat stem rust in the Mississippi Valley now are less frequent and less severe than formerly, because of the eradication of barberries, the changes in cropping systems, and the use of rust-resistant wheats. There is no assurance, however, that other severe outbreaks of stem rust may not occur in the future. The development of an epidemic depends on the presence of viable and virulent inoculum near the growing host and on the environmental factors that favor infection throughout the growing season. With eradication of barberries there has been a great decrease in the quantity of inoculum that once was produced rapidly and in abundance in many local areas. There probably has been a decrease in the diversity of inoculum as well, because many different physiologic races of stem rust may arise on the barberry through hybridization between existing races of *Puccinia graminis*. The growing of resistant varieties of wheat has reduced the

[3] Seed was supplied by Dr. K. S. Quisenberry, Division of Cereal Crops and Diseases, United States Department of Agriculture, cooperating with the Agricultural Experiment Stations of Kansas and Nebraska.

chances for rapid multiplication of inoculum and helped to change the population trends of many of the commonly occurring physiologic races of rust. The classic example of the effect of a new wheat on the rust population is the discovery and rapid increase in prevalence and destructiveness of race 56 following the release and extensive planting of Ceres wheat. Environment plays as great a part in the development of an epidemic of stem rust as does abundant virulent inoculum. Even with early and abundant inoculum of numerous virulent physiologic races of stem rust, the percentage of infection in a variety varies from season to season and even within a season. The year 1942 was exceptionally favorable for development of the artificial epidemic of stem rust, but even in that year conditions were less favorable for rust development early in the season than later.

Race 15 B of *Puccinia graminis tritici* was identified in several stem-rust collections from different parts of the United States, particularly near barberries. It is not one of the most prevalent races of stem rust but is potentially dangerous for the great majority of the wheat varieties and hybrids being bred for various parts of the Mississippi Valley. The varieties now grown in the spring wheat area also are susceptible to race 15 B, but on those hosts many different rust races are competing. Should there be a decided shift to some of the newer hybrids that are susceptible to race 15 B and resistant to the now prevalent races, there would be far less competition among races and more chance for an increase of race 15 B. Many of the breeding materials used as rust-resistant parents are moderately to highly susceptible to race 15 B. Among these are Hope and H 44, most of the Kenya wheats, Iumillo durum, Vernal emmer, and *Triticum timopheevi*, Relatively few wheats are highly resistant to race 15 B and most of these have certain undesirable characters. If biotype B of race 15 once becomes well established in the Mississippi Valley, which is not at all improbable, it may be very destructive to many of the new winter-wheat hybrids, to all the spring-wheat varieties now grown commercially, to most of the newer varieties and hybrids of hard red spring wheat, and to the new durum varieties. Susceptible hosts would be well distributed over the entire central and northern parts of the Mississippi Valley. With an increase in inoculum of race 15 B and with a wide distribution of susceptible wheats, it would be necessary to rely chiefly on environmental factors for the prevention of future stem-rust epidemics until resistance to race 15 B could be incorporated into other desirable wheats.

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INVESTIGATIONS ON THE TRANSMISSION OF BIG VEIN OF LETTUCE

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Big vein of lettuce was first described by Jagger and Chandler.[3] They stated that the disease caused considerable loss to lettuce growers in the Imperial Valley of California. They advanced no theory as to the cause of the disease but reported that it was soil-borne and that partial sterilization with steam or formaldehyde would eliminate it. However, they presented no experimental data on soil transmission.

Big vein has been present in lettuce breeding stocks in the greenhouse and in field plots both at Beltsville, Md., and at Arlington Farm, Va., ever since the lettuce breeding work was begun at Arlington in 1929. Observations made on field trips to the principal lettuce-growing sections of the East during the past several years have indicated that big vein is widespread in the field.

Although the losses in breeding stock resulting from big vein have been small, it is always present in the greenhouse during the cooler parts of the year. Observations by Thompson and Doolittle[4] indicate that there is a temperature above which leaf symptoms are masked. The abnormal appearance of plants affected with the disease interferes with selection for type. Numerous tests of progenies from seed harvested from big-vein plants have yielded no evidence that the disease is seed-borne and its appearance in the greenhouse has suggested both soil transmission and dissemination by insects.

Since Jagger and Chandler reported on the disease in 1934, there have been no published data on the transmission of big vein. The present work began in the fall of 1942. It seemed possible that insect vectors might exist, so arrangements were made with the Bureau of Entomology and Plant Quarantine for cooperative studies and Floyd F. Smith of the Division of Truck Crop and Garden Insect Investigations studied possible insect vectors. Transmission through the soil and by mechanical means was studied by Ross C. Thompson and S. P. Doolittle of the Division of Fruit and Vegetable Crops and Diseases.

LACK OF TRANSMISSION BY MECHANICAL INOCULATION

Up to the present, all attempts to produce big-vein symptoms by mechani-

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[4] Thompson, R. C., and S. P. Doolittle. Influence of temperature on the expression of big vein symptoms in lettuce. Phytopath. 32: 542–544. 1942.

cal inoculation have been unsuccessful. Plants have been inoculated by leaf-rubbing with the use of carborundum powder, and by insertion of crushed tissue in incisions at the juncture of the petiole and stem. Roots were inoculated by dipping, as well as by cutting and crushing the roots while immersed, in juice extracts from big-vein-infected plants. Roots appearing at the opening in the bottom of the pots were inoculated by crushing and also by pricking the juice extract into larger roots. Other inoculations were made by injuring the roots with a spatula driven into the soil during the process. Crushed leaf tissue of big-vein-infected plants also was placed in the soil directly below the roots.

A total of 136 plants were inoculated by one or another of these methods. The inoculated plants were all held for 50 to 60 days at temperatures favorable to the appearance of the disease but no evidence of big vein occurred.

SOIL TRANSMISSION

During the winter of 1941 nearly every lettuce plant in about 300 ten-inch pots in the greenhouse had the typical symptoms of big vein. After the seed from these plants was harvested the soil was removed from the pots and after mixing, was stored in open bins in the field. This soil was used in tests during the winter of 1942–43, to determine whether big vein could be transmitted by this soil and, if so, whether steam sterilization would inactivate the causal agent.

A quantity of this soil that had previously grown big-vein plants was brought to the greenhouse in the late fall of 1942. It was divided, and one-half was steamed for three hours at approximately ten pounds pressure. The other half was left untreated.

In each of 3 tests on the soil transmission of big vein, 50 six-inch pots were filled with untreated soil and 50 with steamed soil. Lettuce sown in the two kinds of soil was transplanted to the respective soils, one plant in each pot. The pots were set on clay dishes to prevent possible contamination of the potted soil from the soil in the benches.

The 3 series were set up and handled in the same way, except that in series 3, the original untreated soil was not used. This series was started December 29, 1942, on the day that series 1 terminated. Soil from the untreated pots in series 1, in all of which big vein had appeared, was used for the untreated part of series 3. This soil was removed from the pots, mixed thoroughly, passed through a ¼-inch mesh screen to remove most of the lettuce roots, and returned to the pots. In other respects the 3 series were comparable except for date of planting.

A summary of the data from the 3 tests is in table 1. The data indicate clearly that big vein of lettuce is soil-transmitted and that the causal agent can be inactivated by steam treatment as stated by Jagger and Chandler.

From 38 to 51 days were required for the first plants to show symptoms of big vein, and a much longer time (88 to 127 days) for some of the plants to develop symptoms. Were it not for the fact that all efforts to transmit

TABLE 1.—*Summarized data from 3 tests on soil transmission of big vein of lettuce, in 1942–1943. Fifty plants in each series, in each soil*

Series I. Started Oct. 2			Series II. Started Nov. 2			Series III. Started Dec. 29		
No. plants showing big vein			No. plants showing big vein			No. plants showing big vein		
Date observed	Sterilized soil	Unsterilized soil	Date observed	Sterilized soil	Unsterilized soil	Date observed	Sterilized soil	Unsterilized soil
Nov. 16	0	1	Dec. 10	0	2	Jan. 22	0	1
Nov. 30	0	6	Dec. 26	0	15	Feb. 5	0	17
Dec. 7	0	10	Jan. 11	0	27	Feb. 15	0	34
Dec. 14	0	21	Jan. 30	1	33	Feb. 27	0	42
Dec. 21	0	36	Feb. 11	0	41	Mar. 8	0	46
Dec. 29	0	50	Mar. 9	0	48	Mar. 15	0	50
Total	0	50		1	48		0	50

the disease by mechanical means were negative, that no root aphids were present in any of the 3 series, and that the plants were kept free of other insects by regular and frequent fumigation of the greenhouse, it might be thought that the disease had been transmitted by some other agent than the soil.

Pryor[5] found that a relatively long time elapsed before big-vein symptoms appeared in his cultures and that some plants required more than 120 days in infested soil before symptoms became evident.

It should also be noted that symptoms did not develop in less time in series 3, in which the soil was used immediately after the removal of big-vein plants, than in the other two series. It is evident that the causal agent may persist for at least one year under field conditions. Further investigations of its longevity in the soil are in progress.

INSECT TRANSMISSION

Some preliminary experiments during the winter of 1941–42 indicated that certain insects might be vectors for big vein. However, the results of

TABLE 2.—*Transmission of big vein of lettuce by insects*

Insect vector	No. insects used	No. trials	No. plants infected[a]	Minimum incubation period,[b] in days
Aphis gossypii	250	1	0/10	.....
Macrosiphum solanifolii	175	2	0/10	.....
Macrosiphum n. sp.[c]	475	4	2/20	33
Myzus circumflexus	325	2	1/15	69
M. persicae	620	5	2/26	43
Trialeurodes vaporaorium	400	2	0/20	.....

[a] Number of plants infected over number exposed.

[b] Days until symptoms appeared.

[c] An undescribed species deposited in collection of Division of Insect Identification, Bureau of Entomology and Plant Quarantine, under T. C. No. 7335.

[5] Pryor, Dean E. The big vein disease of lettuce in relation to soil moisture. Jour. Agr. Res. [U. S.] 68: 1–9. 1944.

these experiments were variable and since adequate precautions had not been taken to prevent infection due to penetration of the roots into unsterilized soil in the bench beneath the pots, the data were considered inconclusive and are not presented. In all later experiments the pots were set on sterilized clay dishes to prevent contamination. After exposure to infective insects the plants were sprayed with a pyrethrum preparation and held in an insect-free greenhouse not previously used for growing lettuce.

The results of tests with five species of aphids and the greenhouse white fly, *Trialeurodes vaporaorium* Westw., are in table 2. No infections occurred in the plants exposed to white flies, to *Aphis gossypii* Glover, or to *Macrosiphum solanifolii* Ashm.; and only one or two infections occurred in the plants exposed to *Macrosiphum* n. sp., *Myzus circumflexus* Buckt., and *M. persicae* Sulz. In these experiments all species of sucking insects found on foliage of lettuce in local greenhouses were used. These included several vectors of a number of other viruses. Methods found successful for the transmission of both persistent and non-persistent viruses were used in these tests.

In later experiments during the spring of 1943, big-vein infection occurred in 8 of 10 plants following a transfer of 15 individual root aphids, *Pemphigus lactucae* (Fitch), from roots of a lettuce plant with symptoms of big vein. The aphids were transferred with a camel's hair brush, free of visible soil particles, to root-tips of healthy lettuce plants through the openings in the bottoms of the pots. The aphids increased rapidly after the transfer.

Uninoculated check plants and groups of plants that had been exposed to aphids and white flies from leaves of big-vein-infected plants were growing in the same greenhouse. After exposure the uninoculated plants were fumigated to eliminate these insects. At the time of the final examination, big-vein symptoms were present in 10 of the 60 uninoculated check plants and in from 40 to 70 per cent of the plants that had been exposed to *Trialeurodes vaporaorium, Myzus persicae,* or *Macrosiphum* n. sp. At this time it was found that the subterranean root aphids, which were unaffected by the fumigations, had become established on the roots of many plants in the above-mentioned groups. The natural spread of these root aphids from the originally inoculated to the other groups of plants seems to be the only logical explanation for the infection occurring in the unexposed checks. The same explanation seemed to hold for the greater portion, if not all, of those exposed to the other aphids and white flies, in view of their previous inefficient performance (Table 2). According to Cutright,[6] the young of *Pemphigus lactucae* depart from the usual behavior of young aphids. Instead of settling about the mother, the newly born microscopic young actively migrate among the soil particles or on the surface for 100 feet or more in search of food.

Investigations on insect transmission of big-vein have been discontinued during the present emergency period. Any further investigations to con-

[6] Cutright, C. R. Subterranean aphids of Ohio. Ohio Agr. Expt. Sta., Bul. 387, pp. 175–238. 1925.

firm the evidence indicating *Pemphigus lactucae* to be a vector of big vein should aim to determine whether the virus is carried internally by the insect and spread by its feeding activities, or merely by external contamination through contact with infected soil. The strong migratory habits of this aphid and its general occurrence on many hosts would tend to increase its importance in the spread of big vein in the field.

SUMMARY

The data presented indicate conclusively that the big-vein disease of lettuce is soil-borne and that its causal agent can be inactivated by steam treatment of the soil.

Under the conditions of these experiments, the symptoms of big vein did not appear in the leaves of any of the lettuce plants until they had grown in infected soil for 5 weeks, and a much longer time (up to 127 days) elapsed before symptoms were observed in some plants.

A total of 136 plants were mechanically inoculated in roots, leaves, and stems, using methods which have been effective in transmitting virus diseases in the case of other hosts. Although the inoculated lettuce plants were held from 50 to 60 days at temperatures favorable for the occurrence of big vein, no symptoms appeared.

The experiments on insect transmission were not extensive enough to justify definite conclusions. However, the results indicate that the root aphid *Pemphigus lactucae* (Fitch) may be a vector for big vein.

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STUDIES OF DODDER TRANSMISSION OF PLANT VIRUSES

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INTRODUCTION

Transmission of the virus of cucumber mosaic by two species of dodder, *Cuscuta subinclusa* Dur. and Hilg. and *C. californica* Choisy, and transmission of curly-top virus by *C. subinclusa* was reported (2) in 1940.

Johnson (8) in 1941 reported the transmission of the viruses of aster yellows, sugar-beet curly top, pea streak, tobacco mosaic, and cucumber mosaic by *Cuscuta campestris* Yuncker.

Kunkel (10, 11, 12, 13), using *Cuscuta campestris*, transmitted the virus of cranberry false blossom to tomato (*Lycopersicon esculentum* Mill.), periwinkles (*Vinca rosea* L.), potato (*Solanum tuberosum* L.), tobacco (*Nicotiana tabacum* L.), and *Nicotiana glutinosa* L.; the virus of peach rosette to tomato; and the virus of potato witches'-broom to sugar beet (*Beta vulgaris* L.).

Transmission of dodder latent-mosaic virus by means of *Cuscuta subinclusa*, *C. campestris*, and *C. californica* was reported by Bennett (3); and Bennett and Munck (4) obtained transmission of the virus of yellow wilt of sugar beet by means of *Cuscuta subinclusa* and *C. campestris*.

It was obvious from the time of discovery of transmission of viruses by dodder that this method of transmission offered possibilities for study of plant virus diseases, especially in connection with determination of the host range of the viruses concerned, not afforded by any other method of attack. Efforts were made, therefore, to compare the efficiency of three species of dodder in the transmission of several viruses representative of recognized virus types. The objective of these experiments was to determine the relationship of different types of viruses to a range of dodder species and to obtain information on the factors involved in transmission.

MATERIALS AND METHODS

Three species of dodder, *Cuscuta subinclusa*, *C. campestris*, and *C. californica*, were studied. The first is characterized by its coarse stems. Several species of green plants, mainly shrubs, growing in low places in southern California, are hosts. The second occurs on alfalfa (*Medicago sativa* L.) and other legumes, and the third is abundant on a number of desert plants of southern California. All three species grow well in the greenhouse and can be colonized on a wide range of experimental plants.

Viruses Tested

Sugar-Beet Curly Top. Several strains of the virus were tested, but a

[1] Indebtedness is acknowledged to Katherine Esau, University of California, College of Agriculture, for the information contained in this paper on the cytology and histology of the haustorium of *Cuscuta subinclusa* and also for figure 3.

strain known to produce severe symptoms on sugar beet was used for the results recorded.

Sugar-Beet Mosaic. The virus used was that which occurs commonly on sugar beets in the vicinity of Riverside, Calif. No strains of this virus have been recognized.

Sugar-Beet Yellow Vein. This virus has not been described but it has occurred each season since 1936 in experimental plots of sugar beets near Riverside, Calif., and is known to occur also in Colorado. It causes distinct yellowing along the main veins of the leaves, dwarfing of the plant (often more marked on one side), and general yellowing of the foliage. It is trans-

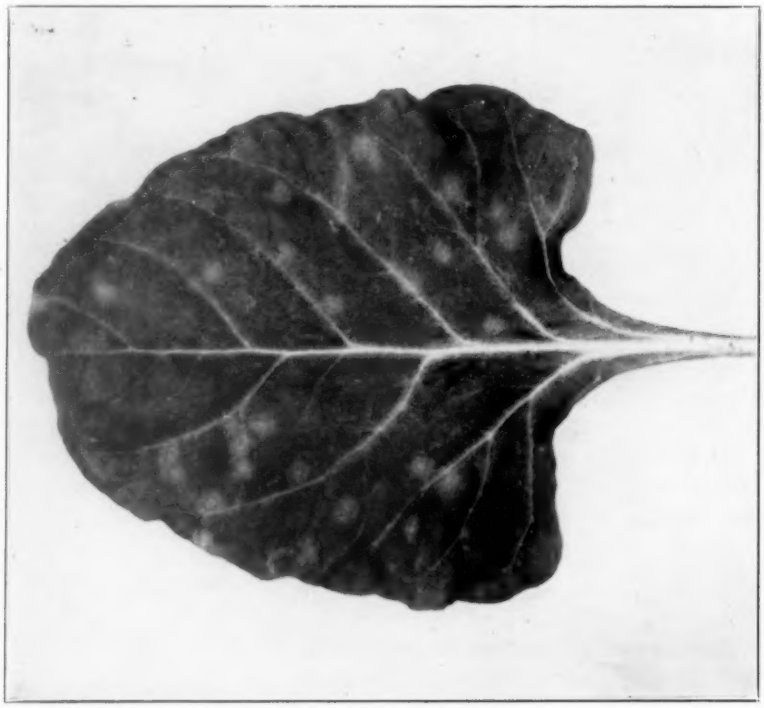

FIG. 1. Leaf of sugar beet, var. S.L. 68, with primary lesions resulting from inoculation with juice from a sugar-beet plant systemically infected by cucumber mosaic.

missible by graft but not by juice inoculation. The vector is unknown, but apparently the virus is not transmissible by the aphid, *Myzus persicae* (Sulz.).

Cucumber Mosaic. The strain of virus used was obtained from sugar beet in the vicinity of Mendota, Calif., where in at least one season it caused severe damage in a relatively large acreage of sugar beets. On leaves of sugar beet yellowish primary lesions (Fig. 1) follow juice inoculation. Systemic infection has not resulted from such lesions, but has resulted from inoculation by *Myzus persicae.*

Tomato Ringspot. The virus was obtained from field tomatoes near Riverside, California. Its relationship to other ringspot viruses of tomato

and tobacco is unknown. It produces necrosis as a primary symptom on both tobacco and tomato. Each type of plant recovers from symptoms.

Dodder Latent Mosaic. This virus was obtained from *Cuscuta californica* and was described in a recent publication (3).

Tobacco Mosaic. The virus (Tobacco virus 1 Johnson) was obtained through courtesy of Wm. N. Takahashi of the University of California, who previously had obtained it from James Johnson, University of Wisconsin.

Mustard mosaic. This virus was obtained from *Brassica adpressa* (Moench) Boiss. near Riverside, Calif., and probably is related to mild-mosaic virus of annual stock described by Tompkins (19). It is common on *Brassica adpressa* and certain other species of mustard in southern California. Systemic infection characterized by mottling occurs on *Nicotiana glutinosa* and necrotic primary lesions on inoculated leaves of Turkish tobacco.

Citrus Psorosis. H. S. Faweett, Citrus Experiment Station, University of California, furnished this virus, which occurs on *Citrus* spp.

Peach Mosaic. L. C. Cochran, Division of Fruit and Vegetable Crops and Diseases, U. S. Department of Agriculture, supplied the virus which came from infected peach (*Prunus persica* (L.) Batsch) trees in Riverside County, California.

Tobacco Etch. The virus was obtained from tomato in a garden in Riverside, Calif. On the basis of induced symptoms and known host range it appears to be related to the tobacco-etch viruses described by Johnson (7). It produces leaflet bending, curling, and mild mottling on tomato. On Turkish tobacco the first affected leaves show yellow veins and abundant fine etching on the upper surface; leaves produced later are mottled, spotted, and stunted. Chlorotic mottling, blossom dropping, and extreme leaf distortion and narrowing are produced on jimson weed (*Datura stramonium* L.); relatively mild mottling, with some leaf puckering, is produced on tolguacha (*D. meteloides* DC.). The thermal inactivation point of the virus lies between 55° and 60° C. It is transmissible by juice inoculation and by the aphid, *Myzus persicae.*

Tomato Spotted Wilt. The virus was obtained from a tomato plant from a garden in Riverside, Calif. In the greenhouse this virus is less severe on tomato and Turkish tobacco than is usual with spotted wilt. Infected plants frequently recover.

Methods of Inoculation

Two methods were employed in making inoculations. Virus-free dodder was usually established on diseased plants and after it had grown sufficiently, stems were trained to healthy plants. Within a few hours the ends of the strands wrapped themselves around stems and petioles of the inoculated plants, and within 48 hours the dodder was sufficiently established on its new host to permit severing its connection with the diseased plant. However, as a rule, strands connecting diseased and healthy plants were maintained for longer periods, usually 7 to 10 days, to permit additional time for viruses to pass from diseased to inoculated plants.

Where it was desired to avoid contact through dodder between the diseased and inoculated plants, strands of dodder several inches long were removed from the diseased plant, placed with the basal ends in bottles filled with water, and the growing tips then were trained around succulent parts of healthy plants. Usually within 2 or 3 days such strands became established on their new host plants.

TRANSMISSION TESTS

Attempts were made to transmit each of the 12 viruses to different species and varieties of plants known to be susceptible to the virus tested. In the

TABLE 1.—*Transmission of viruses by means of Cuscuta subinclusa, C. campestris, and C. californica*

Disease induced by tested virus	Plant inoculated	Number of plants inoculated and infected by means of each species of dodder					
		Cuscuta subinclusa		*Cuscuta campestris*		*Cuscuta californica*	
		Inoc.	Inf.	Inoc.	Inf.	Inoc.	Inf.
Sugar-beet curly top	Sugar beet	141	3	116	12	156	1
	Tobacco, var. Turkish	140	3	40	3	20	2
	Nicotiana glutinosa	28	0	20	0	20	1
	Tomato, var. Riverside	6	0	160	5	27	0
	Sugar beet	140	0	40	0	60	0
Sugar-beet mosaic	Sugar beet	120	0	100	0	120	0
Sugar-beet yellow vein	Sugar beet	20	0	20	0	20	0
Cucumber mosaic	Tobacco, var. Turkish	165	162	30	30	25	23
	Sugar beet	66	3	20	3	29	1
Tomato ringspot	Tobacco, var. Turkish	50	0	15	0	25	0
	Nicotiana glutinosa	20	0	15	0	15	0
Dodder latent mosaic	Sugar beet	50	50	20	20	60	60
	Pokeweed	20	20	65	65	160	160
Tobacco mosaic	Tobacco, var. Turkish	155	3	37	1	48	0
Mustard mosaic	*Brassica adpressa*	93	8	28	2	68	30
Citrus psorosis	*Citrus* sp.	10	0	10	0	.....	.....
	Tobacco, var. Turkish	30	0	.....	.....	.....	.....
Peach mosaic	Tobacco, var. Turkish	20	0	10	0	10	0
Tobacco etch	Tobacco, var. Turkish	23	0	13	0	27	27
Tomato spotted wilt	Tomato, var. Riverside	23	1	60	10	40	1
	Tobacco, var. Turkish	60	0	41	7	41	2

first experiments, *Cuscuta subinclusa* was used exclusively, but later other tests were made with *Cuscuta campestris* and *C. californica*. A partial list of the varieties and species of plants inoculated is in table 1.

The three species of dodder transmitted the virus of dodder latent mosaic to all inoculated plants of sugar beet and pokeweed (*Phytolacca americana* L.); the virus of cucumber mosaic to a high percentage of inoculated plants of Turkish tobacco; and the viruses of sugar-beet curly top and tomato spotted wilt to smaller proportions of inoculated plants. The virus of mustard mosaic was transmitted to a high percentage of plants by *Cuscuta*

californica but to a much lower percentage by the other dodder species. The virus of tobacco etch was transmitted only by *C. californica.*

Transmission of the virus of tobacco mosaic by means of *Cuscuta campestris* was reported by both Johnson (9) and Costa (5). Table 1 shows that a small proportion of plants inoculated with tobacco mosaic virus by means of *Cuscuta subinclusa* and *C. campestris* became infected. However, since accidental infection of Turkish tobacco plants by tobacco-mosaic virus is difficult to avoid in the greenhouse, and since subsequent inoculations (Table 2) in which extreme care was exercised to avoid accidental infection produced no infection, the results do not appear adequate to prove conclusively that tobacco mosaic was transmitted by dodder in these tests.

The incubation period of the viruses in inoculated plants varied considerably. With curly top the incubation period in infected sugar-beet plants was only a few days longer than that in plants inoculated by means of beet leafhoppers. Incubation periods in Turkish tobacco often were very long. In many instances no symptoms of curly top were evident until the inoculated plants began to blossom or fruit. A number of plants inoculated when 3 to 6 inches tall gave no evidence of infection even in the fruiting stage but later developed symptoms on axillary shoots after the plants were cut back to a height of about 8 inches. Incubation periods in these cases ranged from 52 to 80 days. The low percentage of Turkish tobacco plants infected by curly top by means of the 3 species of dodder (Table 1) probably is due in part to failure to hold inoculated plants for periods sufficiently long for symptoms to appear in all cases of infection.

The incubation period of dodder latent mosaic in pokeweed was relatively short, averaging about 7 days as compared to about 10 days required for production of systemic infection from juice inoculation. Also, the incubation period in sugar-beet plants inoculated by means of dodder was relatively short. Frequently the incubation period of the cucumber-mosaic virus in Turkish tobacco was no more than 6 days, which is about the time required for the production of symptoms following juice inoculation. The average incubation period of tobacco-etch virus in Turkish tobacco plants was about 8 days following dodder inoculation compared to about 4 days following juice inoculation.

Marked difference in percentages of transmission of a virus to two different host plants by means of dodder was found in only one instance. Each of the three species of dodder transmitted the cucumber-mosaic virus to high percentages of inoculated plants of Turkish tobacco but transmitted to lower percentages of plants of sugar beet (Table 1). This was true particularly of *Cuscuta subinclusa* and *C. californica.* Why dodder failed to transmit cucumber-mosaic virus to greater percentages of inoculated plants of sugar beet is not known definitely. Although this virus produced numerous primary lesions on leaves of sugar beet following juice inoculation by the rubbing method, systemic infection did not follow in any of more than 200 inoculated plants. Nevertheless, systemic infection is induced more or less

readily by the aphid, *Myzus persicae*. It is possible that to produce systemic infection it is necessary to introduce the virus into tissues of very young leaves. Aphids would be more efficient than dodder in this type of virus introduction.

COMPARISON OF ABILITY OF THREE SPECIES OF DODDER TO TRANSMIT VIRUSES

Early in the course of attempts to transmit viruses by means of dodder it became evident that species of dodder differ in their ability to transmit certain viruses. A series of tests was planned in which the three species were tested in parallel experiments using the viruses of sugar-beet curly top, cucumber mosaic, mustard mosaic, tobacco mosaic, dodder latent mosaic, sugar-beet mosaic, tobacco etch, and tomato spotted wilt. The results are in table 2.

TABLE 2.—*Comparison of ability of Cuscuta subinclusa, C. campestris, and C. californica to transmit viruses*

Disease induced by tested virus	Plant to which transfer was made	Number of plants inoculated and infected by means of each species of dodder					
		Cuscuta subinclusa		*Cuscuta campestris*		*Cuscuta californica*	
		Inoc.	Inf.	Inoc.	Inf.	Inoc.	Inf.
Sugar-beet curly top	Sugar beet	80	8	80	39	80	7
Sugar-beet curly top	Tobacco, var. Turkish	80	12	80	6	80	11
Cucumber mosaic	Sugar beet	80	8	80	32	80	19
Cucumber mosaic	Tobacco, var. Turkish	60	60	60	60	60	60
Mustard mosaic	*Brassica adpressa*	100	12	100	13	100	97
Tobacco mosaic	Tobacco, var. Turkish	80	0	80	0	80	0
Dodder latent mosaic	Pokeweed	100	100	100	100	100	100
Sugar-beet mosaic	Sugar beet	80	0	80	0	80	0
Tobacco etch	Tobacco, var. Turkish	40	0	40	0	40	40
Tomato spotted wilt	*Nicotiana glutinosa*	20	0	20	14	20	1

Cuscuta campestris was the most efficient of the three species in transmission of curly-top virus to sugar beet, but there was little evidence of difference in ability to transmit this virus to Turkish tobacco. *Cuscuta campestris* was the most efficient also in transmission of cucumber-mosaic virus to sugar beet and in transmission of spotted-wilt virus to *Nicotiana glutinosa*. *Cuscuta californica*, however, transmitted the virus of mustard mosaic to a much higher percentage of plants of *Brassica adpressa* than did either of the other species. Also, it transmitted the virus of tobacco etch to all inoculated plants of Turkish tobacco; whereas, there was no transmission by either of the other species. Each species failed to transmit the viruses of tobacco mosaic and beet mosaic.

Some of these differences in the ability of the tested species of dodder to transmit viruses are extremely marked, particularly in the case of transmission of mustard mosaic, tomato spotted wilt, and tobacco etch. These results

suggest the possibility that some of the viruses not transmissible by the species of dodder used in these experiments may be transmissible by other species.

TRANSMISSION FROM PLANTS INFECTED BY TWO OR MORE VIRUSES

It is believed that dodder will prove of value in separating components of virus complexes and especially in separating one or more virus components in diseases caused by multiple infection by viruses that are not differentially transmitted by juice inoculation or by insect vectors. Already, dodder has proved useful in demonstrating double infection in a disease previously assumed to be caused by a single virus. Johnson (9) proved that the white-clover mosaic is caused by two viruses, pea-mottle virus and pea-wilt virus, in combination. The first can be separated from a mixture of the two by use of *Cuscuta campestris*.

TABLE 3.—*Transmission of viruses by means of Cuscuta subinclusa from plants infected by two or more viruses*

Virus combination	Plant inoculated	No. plants inoculated	Number of plants infected by indicated virus		
			Cucumber-mosaic virus	Tobacco-mosaic virus	Curly-top virus
Cucumber-mosaic virus plus tobacco-mosaic virus	Tobacco, var. Turkish	40	38	0	.....
Cucumber-mosaic virus plus curly-top virus	Tobacco, var. Turkish	40	40	.....	3
Cucumber-mosaic virus plus curly-top virus	Sugar beet	80	11	.....	10
Cucumber-mosaic virus plus tobacco-mosaic virus plus curly-top virus	Tobacco, var. Turkish	60	60	0	5

Since Turkish tobacco may be infected by a number of viruses in combination, tests were made using this plant as the virus source in transmission of viruses to Turkish tobacco and sugar beet by means of *Cuscuta subinclusa*. The viruses used were tobacco-mosaic virus, cucumber-mosaic virus, and curly-top virus. The combinations used and the results obtained are in table 3.

Cucumber-mosaic virus in all cases was separated from combinations with tobacco-mosaic virus and frequently from combinations with curly-top virus and from combinations of the three viruses. Of 80 small sugar-beet plants inoculated by training dodder from Turkish tobacco plants containing both cucumber-mosaic virus and curly-top virus, 11 were infected by cucumber-mosaic virus and 10 by curly-top virus. Of the infected plants, 9 were infected by cucumber-mosaic virus alone, 8 by the curly-top virus alone, and 2 by both viruses.

In other experiments, each of the three species of dodder was grown on plants infected by the viruses of dodder latent mosaic and cucumber mosaic

in combination. The dodder was then transferred at weekly intervals for 10 weeks on relatively large sugar-beet plants. All beet plants were infected by the virus of dodder latent mosaic, but only 1 plant (inoculated by means of *Cuscuta campestris*) was infected by cucumber-mosaic virus. Tests on Turkish tobacco at the end of 10 weeks showed that virus of cucumber mosaic was still present in each species of dodder. With smaller sugar-beet plants more infection by the virus of cucumber mosaic, especially by means of *Cuscuta campestris*, would be expected.

PERIOD OF ACTIVITY OF VIRUSES IN DODDER ON NONINFECTED HOST PLANTS

Several methods were used to determine the length of time viruses remained active in the three species of dodder when growing on noninfected plants.

Sugar-Beet Curly-Top Virus. Dodder was established on infected plants and trained from these to healthy sugar-beet plants known to be very resistant to infection by means of dodder because of their large size. After 3 to 5 days the strands of dodder connecting diseased and healthy plants were broken. Tests of virus content of dodder on the healthy plants were made at intervals by allowing nonviruliferous beet leafhoppers to feed on the dodder 24 hours, after which the leafhoppers were caged singly on seedling sugar beets for 7 days. Infection in the seedling beet plants indicated presence of virus in the dodder at the time of test. Virus was recovered readily from dodder 2, 3, and 5 days after the connection with curly-top plants was broken, but smaller amounts of infection after 5 days indicated decreased concentration of virus. Small amounts of virus were recovered after 7 to 10 days but no virus was recovered after longer periods. No differences were noted in the time that any one of the three species of dodder was able to retain virus.

The beet plants on which the tested dodder grew were retained in the greenhouse for more than 2 months after the tests, to determine whether any infection was produced by means of dodder. All remained free of disease. It was assumed, therefore, that these plants did not become infected and that the dodder did not pick up virus from them during the test.

Mustard-Mosaic Virus. Dodder was trained directly from diseased plants of mustard (*Brassica adpressa*) to plants of *Nicotiana glauca* Graham, believed to be immune to infection. At intervals strands of dodder were removed from the plants of *N. glauca* and established on healthy mustard plants. The first tests were made 20 days after the dodder was established on *N. glauca*. No infection of mustard was obtained by means of *Cuscuta subinclusa* or *C. campestris*, but 5 of 11 plants to which *C. californica* was transferred became infected. In tests of dodder 40 days after it was established on *Nicotiana glauca* no infection was produced on mustard by any of the three species of dodder.

Cucumber-Mosaic Virus. It has been reported (2) that *Cuscuta subinclusa* retained the virus of cucumber mosaic for long periods when growing

on an immune host. Also, Costa (5) showed that this virus remained active for 4 months in *Cuscuta campestris* growing on an immune host.

Similar results have been obtained when *Cuscuta subinclusa, C. campestris,* and *C. californica* were grown on noninfected or immune plants. Infected colonies of each of the three species of dodder were established on each of 5 large sugar-beet plants known to be very resistant to infection by the virus of cucumber mosaic owing to their size. At intervals of about 2 weeks a strand of each colony was transferred to a new plant. All dodder was removed from the older plants and these were kept for at least 2 months to determine whether they became infected. At intervals during the test and at the end of 3 months the 5 colonies of each species of dodder were tested for presence of virus. In all of these tests the dodder was found to carry virus. With 4 colonies of *Cuscuta subinclusa,* 2 colonies of *C. campestris,* and 5 colonies of *C. californica,* none of the beet plants on which the dodder grew became infected. It is assumed, therefore, that in these instances the dodder was not able to acquire additional virus from the host plants but that the virus remained active in dodder under these conditions for 3 months.

In other tests the three species of dodder contained cucumber-mosaic virus after growing 40 days on castor bean (*Ricinus communis* L.); *C. subinclusa* contained virus after growing 30 days on *Citrus* sp.; and *C. californica* contained virus after growing 40 days on *Eriogonum fasciculatum* Benth. These host plants of dodder appear to be immune to cucumber mosaic. The virus of cucumber mosaic remains active in each of the three species of dodder almost indefinitely, and probably each species of dodder is a host of the virus.

Dodder Latent-Mosaic Virus. Cuscuta californica and *C. campestris* from infected pokeweed plants were grown for 4 months on plants of *Eriogonum fasciculatum* without loss of the virus. Tests of the *Eriogonum* plants after the infected dodder was removed revealed no infection, and it is assumed that the virus was maintained for 4 months in these two species of dodder while they were growing on an immune host. It is assumed that all three species of dodder are hosts of this virus.

Tobacco-Mosaic Virus. Strands of each of the three species of dodder were removed from Turkish tobacco plants affected by tobacco mosaic, and placed on sugar-beet plants. Seven days later a strand from each of the beet plants was placed on a healthy plant of Turkish tobacco. Twenty tobacco plants were inoculated by means of each species of dodder. None became infected by mosaic. This result is not surprising in view of the small number of plants infected following direct transfer of dodder from diseased to healthy plants (Tables 1 and 2). Both Johnson (9) and Costa (5) obtained greater amounts of infection through direct transfers of strands of *Cuscuta campestris* from diseased to healthy plants. However, they failed to obtain infection by means of this species of dodder after it had been established on an immune host. It seems probable from these results that the virus of

tobacco mosaic remains active in the tested species of dodder for only a relatively short time.

Sugar-Beet-Mosaic Virus. No virus was recovered by juice inoculation from any of the three species of dodder when growing on diseased plants or after transfer to healthy plants.

Tomato-Ringspot Virus. No virus was recovered from any of the three species of dodder when growing on diseased plants or after transfer to healthy plants, either by juice inoculation to Turkish tobacco or by training dodder to Turkish tobacco and tomato.

Tobacco-Etch Virus. The three species of dodder were trained from infected plants of Turkish tobacco to plants of sugar beet, cucumber, Brussels sprouts (*Brassica oleracea* L., var. *gemmifera* DC.), and *Nicotiana glauca,* all of which appear to be immune to infection. After the dodder was established on the immune hosts it was tested for presence of virus by training strands to healthy plants of Turkish tobacco and by inoculation to Turkish tobacco by means of juice from dodder stems. No infection was obtained from *Cuscuta subinclusa* or *C. campestris* at any time by either method of test. However, inoculations by means of strands of *Cuscuta californica* trained to plants of Turkish tobacco after 2 and 4 weeks on the immune host produced infection; but no infection was produced in tests after 6 weeks. Tests with juice from the plants on which the dodder was growing produced no infection on Turkish tobacco. It is evident that *Cuscuta californica* is able to retain the virus of tobacco etch for considerable periods, although it does not appear to be a host of this virus.

RELATIVE CONCENTRATION OF VIRUS IN DODDER AND HOST PLANT

Attempts were made to compare concentrations of viruses in the three species of dodder with those in the respective infected plants on which the dodder was growing. Methods of estimating relative virus concentrations differ with the virus used, and the accuracy of the results varies with the method selected. However, in all cases, it is believed that the methods used are at least of some value in providing information on the probable relative virus concentrations. The results of these tests are in table 4 and the methods used with each virus are indicated in table 4, footnotes a to h, inclusive. All juice inoculations were made by first sprinkling the leaf with carborundum and then rubbing the surface with a cloth pad saturated with inoculum.

Beet leafhoppers obtained the virus of curly top from *Cuscuta subinclusa* in quantities that appeared equal to those obtained from the infected host plant. Smaller amounts of infection, in most cases, were produced by the leafhoppers that fed on *Cuscuta californica* and *C. campestris,* and it seems probable that the leafhoppers that fed on these species obtained somewhat less virus from these species than from the host plant. This may be due to a difference in feeding of the leafhoppers rather than to a difference in concentration of virus in host and parasite.

TABLE 4.—*Concentration of viruses in different species of dodder compared with those in diseased host plants on which the dodder was growing*

Disease induced by tested virus	Diseased plant on which dodder grew	No. infections produced by inoculations from indicated species of dodder and by inoculations from the host plant on which the dodder was growing															
		Cuscuta subinclusa and diseased host plant						*Cuscuta campestris* and diseased host plant						*Cuscuta californica* and diseased host plant			
		Dodder in trial no.			Host in trial no.			Dodder in trial no.			Host in trial no.			Dodder in trial no.			Host in trial no.		
		1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
Sugar-beet curly top[a]	Sugar beet	20	18	18	17	19	20	17	10	12	14	17	20	19	11	8	20	17	19
Sugar-beet mosaic[b]	Sugar beet	0	0	0	48	50	50	0	0	0	50	50	50	0	0	0	50	50	50
Mustard mosaic[c]	*Brassica adpressa*	0	0	0	16	15	19	0	0	0	18	16	17	4	7	10	18	17	20
Mustard mosaic[d]	*Brassica adpressa*	0	0	0	4	11	7	0	0	0	2	8	7	0	1	3	2	14	5
Dodder latent mosaic[e]	Pokeweed	2	12	5	10	19	12	2	2	0	4	11	1	2	2	1	6	13	4
Tobacco mosaic[f]	Turkish tobacco	0.3	0	0	222	124	101	0	0	0	63	81	97	0	0	0	82	76	70
Cucumber mosaic[g]	Turkish tobacco	6	31	47	7	36	63	0	2	2	14	38	37	11	41	42	10	46	51
Tobacco etch[h]	Turkish tobacco	0	0	0	20	20	20	0	0	0	20	20	20	8	4	5	11	3	6

[a] Figures represent the number of infected sugar-beet seedlings of 20 inoculated by means of individual leafhoppers that acquired virus through a 24-hour feeding period on dodder and host plant, respectively.

[b] Figures represent number of infected sugar-beet plants of 50 inoculated by means of juice from dodder and host plant, respectively.

[c] Figures represent the number of plants of *Brassica adpressa* infected of 20 inoculated with juice of dodder and host plant, respectively.

[d] Figures represent average number of primary lesions produced on each of 3 leaves of each of 5 plants of Turkish tobacco after inoculation with juice from dodder and host plant, respectively.

[e] Figures represent average number of primary lesions produced on 3 half-leaves of each of 4 pokeweed plants inoculated with juice from dodder and host plant, respectively.

[f] Figures represent average number of primary lesions produced on each of 3 leaves of each of 3 plants of *Nicotiana glutinosa* inoculated with juice from dodder and host plant, respectively.

[g] Figures represent average number of primary lesions produced on each of 3 leaves of each of 5 plants of sugar beet inoculated with juice from dodder and host plant, respectively.

[h] Figures represent number of plants of Turkish tobacco infected of 20 inoculated with undiluted juice of *Cuscuta subinclusa* and host plant, and of *C. campestris* and host plant, respectively, and with juice, diluted 1 part juice to 1000 parts water, of *C. californica* and of host plant, respectively.

No infection by the virus of beet mosaic was obtained from juice of any of the species of dodder. It is possible, of course, that juice of dodder may tend to inactivate the virus; however, mixtures of 1 part juice of dodder (*Cuscuta subinclusa*) and 1 part juice of diseased sugar beet, undiluted, and diluted 1 to 10, and 1 to 100 with water, gave approximately the same amount of infection when inoculated into sugar beet as corresponding mixtures in which juice of healthy beet was substituted for juice of dodder.

No infection by the virus of mustard mosaic was obtained by means of juice of either *Cuscuta subinclusa* or *C. campestris*. However, by each of 2 methods of tests *Cuscuta californica* gave evidence of having appreciable concentrations of virus, although these concentrations appeared to be lower than those in the infected mustard plants.

In pokeweed and in the three species of dodder the concentration of the virus of dodder latent mosaic rises to a high level a few days after infection, then drops to a low level which is more or less constant thereafter. In tests of relative concentration of this virus, plants that had been infected several weeks were used. In these plants, in which virus content presumably had become more or less stabilized, the concentration of virus was somewhat lower in each of the three species of dodder than in the host plant.

Very few lesions were produced on inoculated leaves of *Nicotiana glutinosa* with juice from dodder growing on Turkish tobacco plants infected by tobacco mosaic. These and other tests, in which never more than 3 primary lesions were produced on a leaf of *N. glutinosa* by inoculation with juice from dodder, show that little infection can be expected from inoculations using juice of dodder from infected Turkish tobacco plants. Costa (5), however, has shown that the juice of *Cuscuta campestris* has an inhibitory effect on the virus of tobacco mosaic. Whether the juices of *C. subinclusa* and *C. californica* have a similar effect has not been determined but tests in which inoculum was prepared by adding 100 parts juice from *C. subinclusa* to 1 part juice from infected tobacco plants gave approximately the same number of lesions per leaf of *Nicotiana glutinosa* as corresponding mixtures of juice from healthy and diseased tobacco plants. It seems probable that the concentrations of virus in the three species of dodder are low as compared with those of the host plants; whether they are so low as indicated by the results presented in table 4 remains to be determined.

The concentrations of virus of cucumber mosaic in *Cuscuta subinclusa* and *C. californica* were about the same as those in the diseased tobacco plants. However, only small numbers of primary lesions were produced on leaves of sugar beet by inoculation with juice from *Cuscuta campestris*. These results are similar to those obtained by Costa (5) from inoculations with juice of *C. campestris* in which he demonstrated that juice of this species of dodder has an inhibitory action on infection. The smaller numbers of lesions produced on inoculated leaves of sugar beet, therefore, probably are due to an effect of this type rather than to a low concentration of virus. There is no evidence, however, that the juice of *C. subinclusa* or of *C. californica* produces a like inhibitory effect.

No infection was produced on Turkish tobacco by the virus of tobacco etch by juice from *Cuscuta subinclusa* or *C. campestris*. However, juice from *Cuscuta californica* produced infection on all inoculated Turkish tobacco plants. In tests in which juice of *C. californica* was diluted 1 part juice in 500, 1000 (Table 4), 2000, and 5000 parts water, respectively, and used to inoculate plants of Turkish tobacco, approximately the same numbers of plants were infected as with corresponding dilutions of juice from diseased plants of Turkish tobacco, indicating that the concentration of virus in this species of dodder was about equal to that of the infected host plant on which it was growing.

TESTS OF THE ABILITY OF DODDER TO TRANSMIT VIRUSES THROUGH SEEDS

Transmission of the virus of dodder latent mosaic through the seeds of *Cuscuta campestris* was reported earlier (3). This work was followed by

TABLE 5.—*Tests to determine whether viruses are transmitted through seeds of dodder*

Virus	Species of dodder	Plant on which tests were made	No. seeds tested	Seeds found to carry virus	
				Number	*Per cent*
Sugar-beet curly top	*Cuscuta campestris*	Sugar beet	240	0	0
Tomato ringspot	" "	Tobacco	160	0	0
Tobacco mosaic	" "	Tobacco	160	0	0
Cucumber mosaic	" "	Tobacco	480	0	0
Dodder latent mosaic	" "	Pokeweed	1340	66	4.9
Dodder latent mosaic	*C. californica*	Pokeweed	670	16	2.4
Cucumber mosaic	*C. subinclusa*	Pokeweed	240	0	0

further tests involving other viruses and two additional species of dodder.

Of the species of dodder used in these tests only *Cuscuta campestris* produced seeds in the greenhouse. This species was grown on several different types of plants each infected by a different virus and seeds were collected for test. Also, large numbers of seeds of *Cuscuta subinclusa* were collected from plants of *Nicotiana glauca* naturally infected in the field by cucumber mosaic. Seeds of *Cuscuta californica* were collected from plants on the desert shrub, *Eriogonum fasciculatum*, in an area where it was known through previous tests that a high percentage of the dodder plants was infected by the virus of dodder latent mosaic.

Seeds were planted in flats and soon after germination the seedlings were attached to young plants known to be susceptible to the virus for which tests for seed transmission were being made. The viruses used and the results obtained are in table 5. The virus of dodder latent mosaic was transmitted through 4.9 per cent of the seeds of *Cuscuta campestris* and through 2.4 per cent of the seeds of *Cuscuta californica*. Seeds of *Cuscuta campestris* still contained active virus after storage for 12 months at room temperature. None of the other viruses tested was seed transmissible.

VIRUS MOVEMENT AS INFLUENCED BY DODDER

Directional Movement in Stems of Dodder

Much evidence indicates that the movement of viruses in the phloem of plants is largely unidirectional and correlated with the movement of elaborated food materials. The evidence on which this conclusion is based was obtained from green plants in which it is not always possible to determine accurately the direction of food movement at all times. With a parasitic plant such as dodder, movement of food materials must always under normal conditions be unidirectional; that is, from the host plant into the parasite and to its regions of growth and storage. Dodder, therefore, may be better adapted for certain types of studies of the correlation between virus movement and food translocation than are green plants.

Limited studies of the movement of viruses in dodder have been made. In the first of these, virus-free dodder (*Cuscuta subinclusa*) was established on healthy plants of *Nicotiana glutinosa*. With each plant, when the first stem of dodder was about 12 inches long, it was attached to a plant of *Nicotiana glauca* infected by the virus of cucumber mosaic. When a stem of dodder from each of these latter plants was 12 inches long it in turn was attached to a healthy plant of *Nicotiana glutinosa*. The 3 plants were allowed to remain attached to each other by the strands of dodder 6 to 16 days, after which the connecting strands of dodder were broken and the plants of *N. glutinosa* were watched for development of symptoms of mosaic.

Of the 10 plants of *Nicotiana glutinosa* to which the virus-free dodder was attached at the beginning of the experiment, only 1 became infected. Hence in 9 of the 10 plants the virus did not move from the infected plant of *Nicotiana glauca* a distance of 12 inches toward the original dodder-infested plant in periods ranging from 6 to 16 days. On the other hand, all of the plants of *Nicotiana glutinosa* to which dodder became attached after making contact with diseased *Nicotiana glauca* plants became infected. Thus the virus was able to move readily in the direction of growth in the dodder stems, but moved less readily from the diseased plant along the stem of dodder to the original healthy plant on which the dodder was established.

In a second test vigorous stems of *Cuscuta subinclusa* more than 2 feet long growing on Turkish tobacco were selected. Twenty beet leafhoppers carrying the curly-top virus were caged on portions, about 7 inches long, of dodder near its points of attachment to the host plant. Twenty nonviruliferous beet leafhoppers were caged on the distal end of each stem. A section of stem, 12 inches or more in length, separated the areas of feeding of the viruliferous and nonviruliferous leafhoppers. At intervals of 1 hour for the first 6 hours and at irregular intervals thereafter, leafhoppers were removed from the cages at the distal ends of the dodder stems and caged on seedling plants of sugar beet to determine whether the leafhoppers had picked up virus.

Beginning with the third hour in some cases and with the fourth and fifth hour in others, leafhoppers removed from cages at the distal end of

dodder stems transmitted curly top to seedling plants of sugar beet. Evidently, this virus was introduced into the basal end of the dodder stem by the viruliferous leafhoppers feeding there and moved through the stem a distance of 12 inches in 3 hours in sufficient quantity to be picked up by nonviruliferous leafhoppers feeding at the other end of the stem. However, in another set of experiments where the relative positions of the viruliferous and nonviruliferous leafhoppers were reversed and leafhoppers carrying virus were allowed to feed on the distal end of the stem, the virus did not move toward the base of the stem a distance of 12 inches during 48 to 72 hours in quantities sufficient to be picked up and transmitted by the nonviruliferous leafhoppers. These results prove conclusively that the virus of curly top moves rapidly outward from the basal portion of a dodder stem in the direction of food flow, but there is no evidence of movement in the opposite direction or against the flow of food materials in periods of 2 to 3 days. This is strong additional evidence of the dependence of virus on food transport for rapid movement through the phloem.

Influence of Dodder on the Movement of Viruses from Inoculated to Noninoculated Shoots in Multiple-Crown Beets

Vigorous growth of dodder on a single infested shoot of a host plant might result in a deficit of food material in that shoot and a resultant movement of food materials from other parts of the plant into the parasitized shoot. The following experiment was made to test this theory.

Roots of sugar beet about 2 inches in diameter and about 8 inches long were split into three parts beginning at the crown and extending downward about 6 inches. They were then potted with tops equally spaced about 6 inches apart. In this manner plants, each with 3 vigorously growing tops all connected at the base of the main root, were obtained. One shoot of each plant was inoculated with a virus, dodder (*Cuscuta subinclusa*) was trained to a second shoot, and the third was retained as a control. Sugar-beet curly-top virus and sugar-beet-mosaic virus were used in the inoculations. Until symptoms of disease developed on the dodder-infested shoot, the dodder was permitted to develop unimpeded except for such pruning as was required to prevent contact with the other two beet shoots and to avoid excessive injury to the infested shoot. After symptoms of disease appeared on the dodder-infested shoot most of the dodder was removed and that which remained was kept severely pruned to avoid markedly interfering with growth of the shoot. One of the plants inoculated with curly top is shown in figure 2, and a summary of the results of these tests is in table 6.

Curly-top virus moved into shoots parasitized by dodder and produced symptoms of disease there in considerably less time than it moved into the control shoots. However, the average period for production of symptoms on the parasitized shoots varied from 32 to 46 days and was considerably longer than that required for production of symptoms on the inoculated shoots. In previous experiments (1) with the same type of beet, in which

VIRUS MOVEMENT AS INFLUENCED BY DODDER

Directional Movement in Stems of Dodder

Much evidence indicates that the movement of viruses in the phloem of plants is largely unidirectional and correlated with the movement of elaborated food materials. The evidence on which this conclusion is based was obtained from green plants in which it is not always possible to determine accurately the direction of food movement at all times. With a parasitic plant such as dodder, movement of food materials must always under normal conditions be unidirectional; that is, from the host plant into the parasite and to its regions of growth and storage. Dodder, therefore, may be better adapted for certain types of studies of the correlation between virus movement and food translocation than are green plants.

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FIG. 2. Sugar-beet plant with 3 shoots on the same root system. A, Shoot inoculated with curly top by means of beet leafhoppers; B, shoot infested with *Cuscuta subinclusa* when A was inoculated; C, control. After inoculation symptoms appeared on the inoculated shoot after 13 days, on the dodder-infested shoot after 38 days, and on the control after 176 days. Plant photographed 100 days after inoculation.

TABLE 6.—*Effect of growth of dodder on movement of viruses into noninoculated shoots of sugar-beet plants split to form three separate crowns all connected through the basal part of the root*

Experiment no.	Disease induced by virus used in test	No. plants tested	Average no. days for appearance of symptoms on indicated shoot		
			Inoculated shoot	Dodder-infested shoot	Check shoot
1	Sugar-beet curly top	10	16	38	113
2	do	4	12	35	127
3	do	3	13	32	153
4	do	5	18	46	139
5	Sugar-beet mosaic	5	13	32	57
6	do	6	8	30	32
7[a]	Sugar-beet curly top	5	17	33	149
	Sugar-beet mosaic	5	12	34	41

[a] The inoculated shoot was infected with the viruses of both sugar-beet curly top and sugar-beet mosaic.

one shoot was defoliated and placed in the dark at the time another shoot was inoculated, symptoms of curly top appeared on the inoculated shoot and on the defoliated or darkened shoot at about the same time. It appears, therefore, that although the dodder greatly accelerated the rate of invasion of a parasitized shoot by the curly-top virus, growth of dodder was not so effective in this respect as removal of all foliage or exclusion of light. This probably is due to the fact that several days are required for dodder to become established sufficiently to create a food deficit in the parasitized shoot and thus induce movement of material, including virus, from the infected shoot.

The average period required for the beet-mosaic virus to move into the parasitized shoot and produce symptoms was of the same order as that required for a similar movement of curly-top virus. However, the beet-mosaic virus moved into the check shoot in a much shorter time than did the curly-top virus.

The quicker appearance of symptoms of beet mosaic on the check shoot probably is due to the types of tissue in which movement takes place. Apparently movement of either virus into the check shoot through the phloem can be prevented so long as a continuous flow of food materials out of this shoot is maintained. Since the virus of curly top apparently moves readily only in the phloem, it may be prevented in this way from entering the check shoot for an indefinite period. However, the virus of beet mosaic, in addition to being able to move through the phloem in the direction of food transport, also is able to move slowly, but at a more or less uniform rate, through the parenchyma, probably by diffusion aided by protoplasmic streaming. Average periods of 32 to 57 days required for the production of symptoms of mosaic on the check shoots probably are long enough to enable the virus of beet mosaic to pass into the check shoots by movement through the parenchyma alone. As in the case of the curly-top virus, however, the virus of beet mosaic can be induced to move rapidly through the phloem into noninoculated shoots by defoliating the shoots or by placing them in the dark.

MECHANICS OF TRANSMISSION OF VIRUSES BY DODDER

Some viruses, such as that of dodder latent mosaic, were transmitted to all inoculated plants by each of the three species of dodder; whereas, others, such as the virus of beet mosaic, were not transmitted. With certain viruses there is considerable variation among the species of dodder in ability to transmit. For example, the virus of tobacco etch was transmitted to all inoculated plants by *Cuscuta californica* but not by *C. subinclusa* or *C. campestris*. All of the factors responsible for these variations are not clear but it seems probable that the differences may be explained in part on the basis of the anatomical relationships of dodder to the host plant coupled with the relationship of different types of viruses to the tissues of the host plant and to dodder.

Anatomy of the Haustorium and Its Relationship to the Host

Since viruses that are acquired by or are introduced into a plant through the medium of dodder must pass through the haustorium, the anatomy of the haustorium and its relationship to the host plant are of extreme importance in any attempt to visualize the mechanics of acquisition or transmission of viruses by dodder.

The formation of the haustorium of *Cuscuta* was described by Peirce (15) and by Thoday (18). According to these investigators, sucker-like processes arise from the epidermis of the dodder stem and adhere firmly to the host plant. A true haustorium, regarded morphologically as an adventitious root, then begins to develop in the cortex in the region of the pericycle. When this comes in contact with the host numerous surface cells of the haustorium develop into elongated hypha-like strands that penetrate deep into the tissue of the host. In this process host cells are absorbed and the strands of the central portion of the haustorium fuse into a more or less compact tissue. Some of the central strands make contact with the xylem and become differentiated into tracheids; others make contact with the phloem.

All investigators agree there is an unbroken tracheal connection between host and parasite through the mature haustorium. This is easily demonstrated even in free-hand sections in *Cuscuta subinclusa* in sugar beet and tobacco. The character of the connection with the phloem, however, is not so clearly evident. Thoday (18) states that in the species of *Cuscuta* studied by her the hypha-like cells of the parasite enlarged at the tip as they approached sieve tubes of the host and the cell walls became plastic and gelatinous. The cell walls at the tips seemed to disappear and the protoplasm applied itself directly to the wall of the sieve tube of the host usually either over a lateral sieve plate or a sieve field. The strands then developed into sieve tubes with short cells.

In *Cuscuta odorata* on *Pelargonium zonale* (L.) Willd., Schumacher (16) found that the ends of elongated feeding cells made contact with the host sieve tubes and flattened out and elongated in one or both directions parallel to the long axis of the sieve tube. Numerous finger-like processes grew out and firmly clasped the sieve tube. Thin walls of the cells of the parasite were directly attached to the unbroken wall of the sieve tube and no evidence of lateral sieve plates or of plasmodesmata connecting host with parasite was obtained. The feeding strands were not observed to differentiate into sieve tubes in the haustorium. In passage from the sieve tubes of the host to those of the parasite it would be necessary under these conditions for materials to move through the cell wall of the host sieve tube, the cell wall of the feeding cell of the parasite and through parenchyma-like tissue traversing the entire length of the haustorium.

Sections of petioles of sugar beet and stems of *Nicotiana glauca* and Turkish tobacco, parasitized by *Cuscuta subinclusa*, were prepared and studied by Katherine Esau of the University of California. She[3] found

[3] Letter to the writer.

cells in the haustorium that had no nuclei but possessed sieve plates with evident callus cylinders. Like typical sieve-tube elements, these cells contained little chromatic material and had companion cells. These sieve tubes were traced to their union with the long hypha-like cells that in their turn connected with the phloem of the host and formed among the phloem cells the finger-like processes described by Schumacher (16). The method of union between the haustorial cells and the sieve tubes of the host was not ascertained.

Regardless of the technical terminology that should be used to designate the food-transporting tissue throughout the length of the haustorium, it is evident that within the haustorium there is a tissue that functions in a highly efficient manner in the transport of materials from the host to the parasite. *Cuscuta subinclusa* on tobacco sometimes produces a stem elongation of 3 inches or more in 24 hours. This amount of growth would involve the transport of relatively large volumes of material. Schumacher (16), on the basis of growth rates and cross-sectional areas of the haustorium, calculated that materials moved out of the host plant into *Cuscuta odorata* at a rate of from 1.6 to 6.2 mm. per hour where movement was considered to take place through the total cross-sectional area of the haustorium. However, since movement probably takes place through only a limited part of the total cross-sectional area of the haustorium, actual rates may be far in excess of those indicated by these figures.

The haustorium of *Cuscuta subinclusa* makes a close union with the tissues of sugar beet and tobacco. The line of demarcation between host and parasite is evident toward the surface of the host tissue but in the vascular region and in the part immediately outside of the vascular region union is so close that it is difficult, in some cases, to differentiate between cells of the parasite and those of the host.

In *Cuscuta odorata* Schumacher and Halbsguth (17) found that the long, hypha-like, terminal cells that radiate from the haustorium and grow through the parenchyma of the host have what they interpreted as numberless fine plasma threads in their walls that pass through the walls to their outer surfaces. Such threads were lacking, however, in walls of cells that attached themselves directly to the sieve tubes of the host.

In sections of the haustorium of *Cuscuta subinclusa* in sugar beet and tobacco with which a staining technique to show plasmodesmata was employed, Esau[4] found darkly stained granules in linear series traversing the walls of haustorial cells that penetrated into the cortical parenchyma of the host. Students of plasmodesmata commonly interpret such lines as a result of the precipitation of the stain (gentian violet in this instance) in places where cytoplasm was present in the living state. If this interpretation is correct, the granular lines within the walls of the host and parasite cells indicate the position of plasmodesmata in these walls. The arrangement and density of these lines are illustrated in figure 3. They are numerous and

[4] Letter to the writer.

rather uniformly distributed in the haustorial cell walls and are continuous from the lumen of the haustorial cell to the lumen of the invaded host cell. In the parenchyma cells of the host the lines are numerous in some walls, few or absent in others, and often are confined to the pit areas. In figure

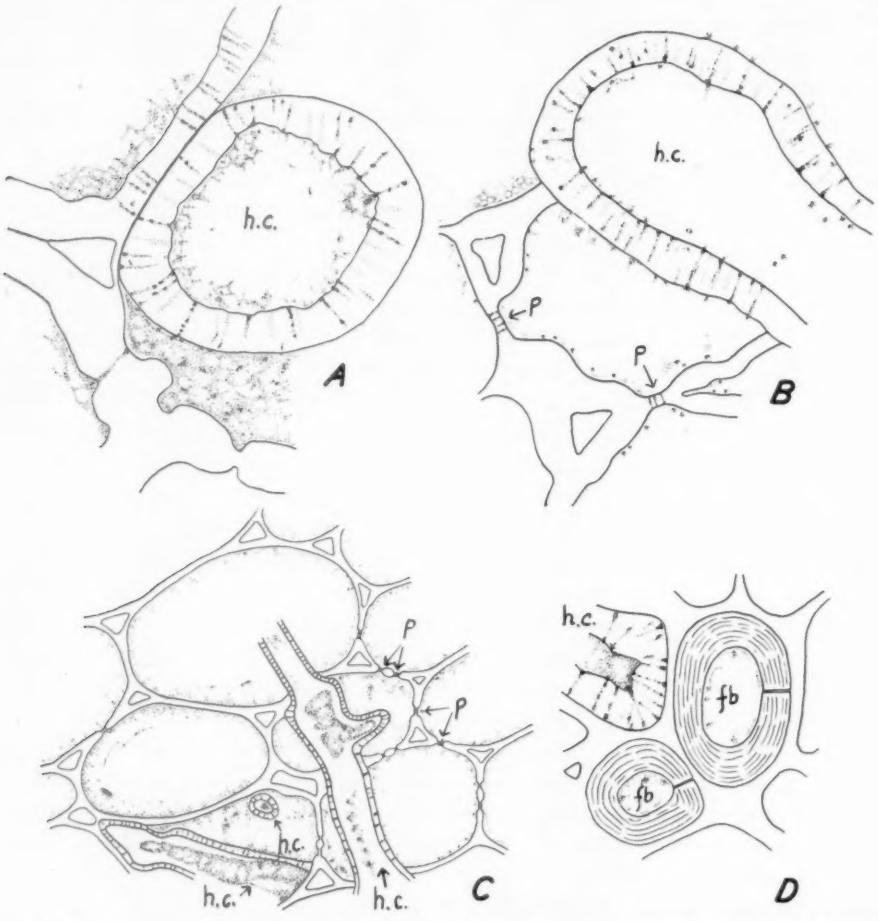

FIG. 3. Free-hand sections of tobacco and sugar-beet stems and petioles showing the relation of the haustorial cells of *Cuscuta subinclusa* to the cells of the host. Plasmodesmata are in the haustorial cells and in the walls of the host plants, either scattered throughout the wall (A) or concentrated in the pit areas (B and C at p). A, Haustorial cell in transverse section adjacent to a wall of a parenchyma cell of *Nicotiana tabacum;* B, end of a haustorial cell and a portion of a parenchyma cell of *Nicotiana glauca;* C, sugar-beet parenchyma with sections of three haustorial cells; D, branch of haustorial cell adpressed to the walls of a primary-phloem fiber (fb) of *Nicotiana glauca,* secondary walls of the fibers marked by concentric lines. fb, fiber; h.c., haustorial cell; p., pit. A, B, and D × 730; C × 163. Sections and drawings by Katherine Esau.

3, A, lines passing through the walls of the outermost cell of the haustorium meet similar lines traversing the wall of the adjacent host cell.

All haustoria illustrated in figure 3 occurred within the parenchyma tissues of the host. In the walls of the ultimate ramifications of the haus-

torial cells within the phloem of the hosts no plasmodesmata were identified with certainty.

If these darkly-stained lines traversing the walls of the peripheral cells of the haustorium of *Cuscuta subinclusa* and similar structures in the walls of hypha-like strands of *Cuscuta odorata* represent plasmodesmata, it is evident that the protoplasm of the parasite may provide direct contact with protoplasm of the host through the medium of these protoplasmic strands passing through the cell walls. This is of special significance for the transmission of certain types of viruses by dodder, since there is strong evidence pointing to plasmodesmata as the avenues of virus movement in the passage of viruses from cell to cell in the parenchyma of flowering plants.

Movement of Viruses into Dodder

If food movement is the result of flow of liquid content of the phloem from points of higher pressure to those of lower tension as suggested by Münch (14) and by Crafts (6), any material, including virus, not filtered out by passage of the liquid through intervening cell walls or membranes, would pass readily from the phloem of diseased plants into dodder. The concentration of virus in the phloem of dodder soon would equal or surpass that in the phloem of the host if not rapidly inactivated in the phloem of dodder. The concentration of virus in dodder, therefore, probably would be influenced decidedly by the concentration of virus in the phloem of the host plant.

It may be assumed with considerable certainty that the virus of curly top is closely limited to the phloem of its host plants. This may be true also for most of the "yellows" type of viruses that produce disturbances in the phloem but which do not produce mottling, rings, local lesions, or other types of disturbances that indicate a direct effect on the parenchyma. Viruses causing mottling, various types of lesions, and other types of disturbances in parenchyma, on the other hand, occur in both phloem and parenchyma, although there is some evidence that the concentration of such viruses in the phloem, in some cases at least, may be low compared with that in the parenchyma.

On the basis of this reasoning it would be expected that phloem-limited viruses, as a rule, would pass into dodder in relatively high concentrations. Also it would be expected that mosaic viruses would move into dodder in lower concentrations and, if unable to invade parenchyma of dodder and accumulate concentrations of virus there, often would be found in low concentrations in dodder, especially if the rate of inactivation in dodder were relatively high. This may account for the very low concentration or absence of the virus of tobacco mosaic, beet mosaic, and tomato ringspot in dodder growing on diseased plants. In cases, however, where dodder is a host of the invading virus, such viruses would multiply in the dodder and reach a high concentration. In certain cases where dodder is not a host of the virus it may be that appreciable concentrations of virus are reached in dodder by invasion of parenchyma and temporary storage in this type of tissue.

It is possible also that mosaic viruses may enter dodder through the parenchyma contacts of the haustorium with the host; but, since this type of entry involves movement through a considerable amount of parenchyma tissue in which movement is slow, it would not be expected to influence appreciably the concentration of virus found in the dodder.

Movement of Viruses Out of Dodder

There appear to be three ways by which it is possible for viruses to move from dodder into healthy host plants and produce infection: (a) The virus may move through the phloem or food transport cells of the haustorium counter to the prevailing direction of food transport and become established in the host plant where it would be able to multiply and produce systemic infection. (b) There is some question as to whether or not the tips of some of the hypha-like strands of the haustorium that penetrate the host cells have well-defined cell walls. If the growing tips of these structures are naked protoplasm and susceptible to infection, virus may pass by way of these directly from the protoplasm of the parasite into the protoplasm of the host and continue to move through the parenchyma tissue of the host until a systemic infection is produced. (c) If the structures observed traversing the walls of haustorial cells of *Cuscuta subinclusa* and *C. odorata* are plasmodesmata it would seem possible for any virus able to invade the outer parenchyma cells of the haustorium to move along these protoplasmic strands and enter the host protoplasm either through similar strands penetrating the walls of adjacent host cells or by passage into host protoplasm applied to the outer surface of walls of the invading hypha-like cells. Invasion of the host parenchyma and eventually of the entire plant could then take place.

The path taken by a virus in passing from dodder to a host plant, therefore, would be determined largely by its relationship to the different tissues of dodder and the host plant. It would be expected that the phloem-restricted (yellows type) viruses and parenchyma-inhabiting (mosaic type) viruses would have rather marked differences in relation to their transmissibility by dodder.

Viruses of the Yellows Type. The path available for passage of phloem-limited viruses would seem to be largely or wholly restricted to specialized transport cells (sieve tubes?) of the haustorium. At what stage in haustorial development this pathway would become available for virus movement into the host is problematical. If the hypha-like cells of the haustorium that later develop into food transport cells are subject to invasion by virus, it is possible that infection of the host may occur at the time contact between these hypha-like cells and the sieve tubes of the host is made, or soon after. If virus is unable to pass into these young haustorial cells before they develop into transport cells, as seems more likely, and infection is delayed until movement of food materials out of the host begins, infection then would involve movement of virus out of dodder into the host plant counter to the

general direction of movement of elaborated food materials. However, this movement would be through a very short distance, for when dodder is trained from a diseased to a healthy plant the virus continues to pass, for a time at least, from the diseased plant along the stem of dodder past the new haustoria developing in the healthy plant. A virus such as that of curly top, moving from the diseased plant, would pass through the phloem in the strands of dodder within a millimeter, more or less, of the phloem of the healthy plant. Moreover, so long as the virus retained its activity it would be present in the phloem of dodder only a short distance from the phloem of the new host. The fact that virus of curly top did not bridge this short distance and produce infection in dodder-inoculated plants more often than was found is evidence of a high degree of efficiency on the part of dodder in maintaining a condition of continuous flow of material out of the host plant.

The frequency with which such viruses are able to pass through the haustorium and enter the host plant probably depends on concentration of virus in the dodder, duration of period of activity of virus in the phloem of dodder, and perhaps on other factors such as temporary slight reversals of food flow between dodder and host. Since phloem-limited viruses probably occur in relatively high concentrations in the phloem of their hosts, it would be expected that they would enter dodder in relatively high concentrations. All such viruses that retain their activity for appreciable periods of time in the phloem of dodder should be transmissible; but on the basis of the evidence regarding the difficulty of movement of virus through the haustorium counter to the direction of food flow they should be transmissible, as a rule, to relatively low percentages of the inoculated plants except in cases where dodder is a host of the virus, in which case high percentages of infection should be obtained, but perhaps only after longer periods of contact between the infected dodder and healthy host plant.

These conclusions are supported by the available results of tests with *Cuscuta campestris* (Table 7). Of the 19 viruses listed, 7 are classified as belonging to the yellows group and all of these are transmissible by *Cuscuta campestris.* Four of the 6 viruses on which data are available, were transmitted to 26 per cent of inoculated plants or less. Dodder has not been shown to be a host of any of these 4 viruses. A high percentage of infection was obtained with each of the 2 remaining viruses. Of these latter, Costa (5) has shown that the virus of cranberry false blossom multiplies in *Cuscuta campestris;* and the high percentage of plants infected by potato witches'-broom virus suggests that *C. campestris* also may either be a host of this virus or able to retain active virus for a long time. On the basis of these results it may be expected that many other viruses of the yellows type will prove to be transmissible by dodder.

Viruses of the Mosaic Type. If present in comparable concentrations in the phloem of dodder, it would be expected that mosaic viruses would be able to produce infection as readily as yellows viruses by moving through food transport cells of the haustorium into the host counter to the direction of

movement of materials into the parasite. However, since there is evidence that some of the mosaic viruses occur in low concentrations in the phloem of their host plants, it may be that as a rule these viruses are picked up in low concentrations by dodder. Furthermore, if the phloem of normal host plants is an unfavorable medium for such viruses, the phloem of dodder should prove to be an even more unfavorable medium, and after passage into dodder the initial low concentration of virus probably would be reduced still further by inactivation. Thus mosaic viruses not only may enter the phloem

TABLE 7.—*Summary of information available on transmission of viruses by Cuscuta campestris*

Disease induced by tested virus	Plant inoculated	No. plants inoculated	Plants infected		Authority[a]
			Number	*Per cent*	
Yellows type:					
Sugar-beet curly top	Sugar beet	196	51	26	.....
Sugar-beet yellow wilt	Sugar beet	17	4	24	(4)
Aster yellows	Aster	50	4	8	(9)
Tomato bushy stunt	Tomato	20	5	25	(9)
Cranberry false blossom	Tomato	93	69	74	(5)
Peach rosette	Tomato	Not stated	+[b]	?	(12)
Potato witches'-broom	*Vinca rosea*	48	42	82	(13)
Mosaic type:					
Sugar-beet mosaic	Sugar beet	180	0	0	.....
Cucumber mosaic	Tobacco, var. Turkish	90	90	100	.....
Mustard mosaic	*Brassica adpressa*	128	15	12	.....
Tobacco mosaic	Tobacco, var. Turkish	117	1[c]	1	.....
Tomato ringspot	Tobacco, var. Turkish	40	0	0	.....
Tobacco etch	Tobacco, var. Turkish	40	0	0	.....
Dodder latent mosaic	Pokeweed	120	120	100	.....
Tomato spotted wilt	Tobacco, var. Turkish	41	7	17[d]	.....
Citrus psorosis	Citrus sp. seedlings	10	0	0	.....
Tobacco ringspot	Tobacco, var. Turkish	32	0	0	(9)
Pea wilt	*Medicago lupulina* L.	8	5	62	(9)
Pea mottle	*Medicago lupulina*	8	0	0	(9)

[a] Where authority is not indicated, results are those reported in this publication in tables 1 and 2.

[b] Plus sign indicates that infection was obtained but number of infected plants was not stated.

[c] However, Johnson (9) reported infection in 13 of 26 Turkish tobacco plants and Costa (5) reported infection in 5 of 31 tomato plants inoculated by means of *Cuscuta campestris*.

[d] In limited tests a higher percentage of infection was obtained on *Nicotiana glutinosa* (Table 2).

of dodder in low concentrations but may tend also to be lost rapidly from the phloem, possibly in some cases before they pass through considerable lengths of stems of dodder. For these reasons it may be that infection by mosaic viruses by passage from dodder to the host plant through the food transport cells of the haustorium would occur less often than with yellows viruses that may occur in higher concentrations in the phloem and be better adapted to survive in this type of tissue.

However, mosaic viruses invade the parenchyma of their host plants and reach relatively high concentrations in such tissue, and it is probable that

transmission of mosaic viruses to high percentages of inoculated plants will be found associated with ability of these viruses to invade parenchyma of dodder. Invasion of parenchyma would enable virus to move through the haustorial tissues to the peripheral cells of the haustorium from which it would be able to move through the plasmodesmata traversing the walls of cells in or adjacent to those of the host and invade the protoplasm of the host plant. As already suggested, it is possible also that if the tips of young invading hypha-like cells of the haustorium are devoid of cell walls and contain virus, infection by mosaic viruses may take place by direct passage of virus from the invading protoplasm to that of the host.

In view of the evidence indicating low concentration of mosaic viruses in the phloem of dodder, occurrence of such a virus in relatively high concentration in the juice of dodder should be evidence of its ability to invade parenchyma of dodder. Therefore, all mosaic viruses that occur in relatively high concentrations in expressed juice of dodder should be transmissible by dodder to high percentages of inoculated plants.

These conclusions are supported by information on transmission of parenchyma-inhabiting viruses by means of *Cuscuta campestris* presented in table 7. Of the 12 viruses listed, 2 were transmitted to 100 per cent of inoculated plants, 1 to 62 per cent of inoculated plants, 3 to small percentages of inoculated plants, and 6 to none of the inoculated plants. *Cuscuta campestris* appears to be a host of both of the viruses transmitted to 100 per cent of inoculated plants. No information is available regarding the relationship to *C. campestris* of the virus transmitted to 62 per cent of inoculated plants. There is no evidence that the viruses of any of the other diseases transmitted to low percentages of inoculated plants or nontransmissible occur in dodder in any appreciable concentration. Also, with *Cuscuta subinclusa* and *C. californica* there is a marked correlation between indicated concentration of virus in dodder (Table 4) and percentage infection produced by means of dodder on inoculated plants (Tables 1 and 2). Strong additional support for these concepts is afforded by the fact that *Cuscuta californica* had relatively high concentrations of virus of tobacco etch, transmitted the virus from diseased to all inoculated plants of Turkish tobacco, and retained virus 4 weeks while growing on immune hosts; whereas, in parallel tests no virus was recovered from either *Cuscuta subinclusa* or *C. campestris* and no infection was obtained by means of either of these species.

With the mosaic type of virus, therefore, it may be expected that as a rule transmission by means of dodder will prove to be either very high or very low or absent. Those viruses that are able to utilize dodder as a host or that are able to invade the parenchyma of dodder and retain their activity for considerable periods in such tissue will be transmissible to a high percentage of inoculated plants; whereas, those not able to utilize dodder as a host or to invade the parenchyma of dodder will be, as a rule, either not transmissible or transmitted sporadically.

SUMMARY

Transmission of 12 plant viruses by three species of dodder, *Cuscuta subinclusa*, *C. campestris*, and *C. californica*, was studied. The viruses of dodder latent mosaic and cucumber mosaic were transmitted to high percentages of inoculated plants by each of the three species of dodder. The viruses of curly top and spotted wilt were transmitted to smaller proportions of inoculated plants. The virus of mustard mosaic was transmitted to high percentages of inoculated plants by *Cuscuta californica* but to low percentages by the other two dodder species. The virus of tobacco etch was transmitted to all inoculated Turkish-tobacco plants but was not transmitted by either of the other species. No transmission of the virus of tobacco mosaic was obtained with *Cuscuta californica* but a very low percentage of Turkish tobacco plants inoculated by means of the other two species became infected. No transmission was obtained of the viruses of sugar-beet mosaic, sugar-beet yellow vein, tomato ringspot, citrus psorosis, or peach mosaic by any of the species of dodder.

In tests to determine longevity of viruses in dodder growing on noninfected host plants, the viruses of cucumber mosaic and dodder latent mosaic were active in the three species of dodder after periods of 1 to 4 months. The virus of curly top was not recovered in periods longer than 10 days. The virus of mustard mosaic was active in *Cuscuta californica* after 20 days but not after 40 days; apparently it was lost from *C. subinclusa* and *C. campestris* in shorter periods. The virus of tobacco etch persisted in *Cuscuta californica* for periods of 2 to 4 weeks but was not recovered from the other two species.

Tests of relative concentration of virus in dodder and infected plants on which the dodder was growing indicated that the concentrations of curly top virus in dodder were about equal to or slightly less than those in diseased sugar beet. Concentrations of the virus of dodder latent mosaic were somewhat lower than in diseased pokeweed plants. Concentrations of cucumber-mosaic virus were about equal to those of the host in *Cuscuta subinclusa* and *C. californica*, but little virus was recovered from juice of *C. campestris*, possibly because of inhibitory effects of juice of this species. Concentrations of the virus of mustard mosaic in *Cuscuta californica* were considerably lower than those of the host; no virus was recovered from *C. campestris* or *C. subinclusa*. Concentrations of virus of tobacco etch appeared to be as high in *Cuscuta californica* as in the host but no virus was recovered from the other two species of dodder. Concentration of the virus of tobacco mosaic appeared to be low in each species of dodder as compared with the host. No virus was recovered from dodder growing on plants infected by sugar-beet mosaic, sugar-beet yellow vein, or tomato ringspot.

The virus of dodder latent mosaic was transmitted through 2.4 per cent of the seeds of *Cuscuta californica* and through 4.9 per cent of the seeds of *C. campestris*. The virus was active in seeds of the latter species after a storage period of 1 year.

The movement of the viruses of curly top and cucumber mosaic in dodder stems was much more rapid toward growing points and away from the host than in the opposite direction. Growth of dodder on one shoot of beet plants with three shoots on a single root system induced movement of the curly-top virus from an inoculated shoot in periods of from 32 to 46 days; whereas, check shoots remained free of symptoms for periods of 113 to 149 days.

The tracheal elements of dodder make direct contact with those of the host through the haustorium. There is disagreement regarding the nature of the cells uniting the phloem of the parasite with that of the host but it is evident that food materials move rapidly through these cells and that they function as phloem. The walls of certain outer cells of the haustorium are traversed by lines that appear to be plasmodesmata. These in some cases appear to join with similar lines in walls of adjacent host cells.

Viruses appear to be acquired by dodder mainly by movement from the host into the parasite through the phloem with the food materials. Infection probably takes place: (a) by movement of virus from the phloem of dodder through the haustorium into the phloem of the host counter to the prevailing direction of food movement, and (b) by passage from the parenchyma of the haustorium into that of the host through plasmodesmatal strands or from naked protoplasm of invading hypha-like cells. It is suggested that infection by yellows-type viruses may be effected by the first method and that infection by mosaic-type viruses may be effected chiefly by the second method. An analysis of the available information on transmission of viruses by *Cuscuta campestris* shows that all tested viruses classified as yellows types are transmitted but, in general, to low percentages of plants; whereas, relatively few of the mosaic-type viruses are transmissible but those that are transmitted in general are transmitted either to a very high or to a very low percentage of inoculated plants.

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REPORT OF THE TWENTY-SEVENTH ANNUAL MEETING OF THE PACIFIC DIVISION OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

The 27th annual meeting of the Pacific Division of the American Phytopathological Society was held at Oregon State College, Corvallis, June 26 to 28, 1944. Thirty-two members and 12 non-members from Washington, Oregon, California, Idaho, Nevada, and Washington, D. C., attended the scientific sessions and a lesser number attended the two field trips. Twenty-two papers were presented. At the business meeting it was voted to purchase three $100 War Bonds in the name of the American Phytopathological Society from the funds of the Pacific Division.

Officers for 1945 were elected as follows: President: B. L. Richards; Vice-President: H. R. McLarty; Secretary-Treasurer: Geo. W. Fischer; Councilor: L. D. Leach; Representative, Pacific Division A.A.A.S.: L. D. Leach.

Abstracts of papers presented follow.

C. E. YARWOOD,
Secretary-Treasurer.

Further Evidence of Pollen Dissemination of Walnut Blight. P. A. ARK. In the spring of 1944, many recently pollinated nuts (3 to 5 mm. in size) in a commercial walnut orchard were completely blighted by *Phytomonas juglandis.* No foliar blight and hold-over lesions occurred on the majority of trees examined. This condition prevailed on both Bordeaux-sprayed and unsprayed trees. Wash water from apparently healthy leaves and nuts from trees in the blighted orchard yielded virulent cultures of *P. juglandis.* Young Northern California black walnut trees, although planted 25 to 50 feet from blighted Payne trees had numerous leaf infections from the tree top to the ground line. However, no cankers or blighted dormant buds could be detected on these trees. In the absence of hold-over lesions, it is assumed the disease was induced by walnut pollen grains abundantly present on the leaves. Since many nuts are initially blighted at the apical end, and because some well-separated, young, non-bearing trees develop foliar blight in the absence of overwintering foci of infection, it is believed the high incidence of the disease this spring may be attributed to contaminated wind-borne walnut pollen, originating in partially blighted catkins of the highly susceptible Payne variety.

Bacterial Blight of Purple Vetch Caused by Phytomonas pisi. P. A. ARK. In a field of several acres in Sonoma County, California, purple vetch (*Vicia atropurpurea* Desf.) and Canadian field peas (*Pisum sativum* L. var. *arvense*) were found severely blighted by *Phytomonas pisi.* In many cases the disease on the vetch originated at the middle of the stem, spread rapidly toward the base, and quickly killed the plant. Occasionally only the tips of the plants were blighted, and the progress of the disease probably was checked by an increase in temperature. Pea plants developed blight lesions with isolates from both hosts, and successful cross-inoculation was obtained in the greenhouse.

Phytophthora Root Rot of Guayule. ALVIN J. BRAUN. A root rot caused by *Phytophthora drechsleri* Tucker has been observed in many irrigated field plantings of guayule in California, especially on the heavy, poorly drained soils. The greatest loss occurs on the clay soils of the Tracy-Newman district where root rot was observed in 22 of the 25 fields surveyed in September, 1943. Losses of 5 per cent or more of the plants were recorded for 625 of the 3745 acres included in the survey. When the lesion girdles the tap root the plant wilts rapidly, the leaves turn gray, dry, and usually remain firmly attached to the plant for the remainder of the season. The root lesions are black, sunken, and firm, the upper margin usually being 2 to 6 inches below the ground line. Field observations and temperature tank studies show that soil temperatures above 60° F. are necessary for infection. Soil moisture in excess of the field capacity for extended periods is particularly conducive to the development of root rot. This condition is more readily attained on the heavy, poorly drained soils and on soils underlain with a claypan or hardpan. Control is effected by avoiding soil moisture conditions favorable to infection at times when the soil temperature at the 6-inch depth is above 65° F.

Black Streak, a Bacterial Disease of Sugar Beet in the Pacific Northwest. EUBANKS CARSNER. A bacterial disease characterized by black or dark brown lesions on leaf blades, petioles, and seed stalks of sugar beet has been observed near Gazelle, Ft. Jones, and Montague in northern California, in the vicinity of Mt. Vernon in western Washington, and for several years and in many places in the Willamette and Rogue River valleys of Oregon. The causal organism apparently enters through hydathodes and through wounds.

Frequently it invades tissue injured by downy mildew. Field observations suggest that the causal organism may be seed borne. Sugar beet varieties vary in susceptibility to black streak, but in no field observed has the injury been a major factor affecting seed yield. The causal bacterium is probably the same as that described as *Bacterium aptatum* by Nellie A. Brown and Clara O. Jamieson (Jour. Agr. Res. 1: 189–210. 1913).

The "Complex Concept" of the Peach Mosaic and Certain Other Stone Fruit Viruses. L. C. COCHRAN. Several years' study has shown that peach mosaic virus from individual sources produces uniform symptoms on Hale peach, but the same virus from separate sources varies extensively in intensity and type of symptoms. Viruses from different sources were previously arbitrarily divided according to effect into severe, medium, and mild categories and were called strains. Further studies indicate the existence of infinite categories and, given enough sources, these overlap to form a symptom gradient in which no definable strains can be segregated. When peach mosaic virus from various sources is placed in apricot and certain other stone fruits, a severe to symptomless gradient results which may or may not agree with the severity in peach. Mild to severe symptom gradients are also known for ring spot, asteroid spot, and certain of the cherry viruses. If certain viruses are groups there is need for group terminology. Following a suggestion of C. W. Bennett, it is proposed that the term *strain* be reserved for variants that are sufficiently definable to be recovered from nature and identified. It is further proposed that the term *form* or *isolate* with appropriate descriptive adjectives be used for viruses from individual sources which vary widely in symptom expression. Collectively such viruses may be referred to as *complexes*.

Some Host-tissue Relationships of the Peach Mosaic Virus. L. C. COCHRAN and JOHN L. RUE. Evidence that the peach mosaic virus invades all living parts of the peach tree except seeds has been obtained through transmission with infected leaf, fruit, bark, and wood tissue. Inoculations were made by placing the infected tissue under the bark in T cuts, as in budding. Some leaf sections were green 75 days after inoculation and appeared to have united with the wound callus. Some of the fruit sections remained firm and green 25 days after inoculation. Transmission was accomplished with green, hard ripe, and with firm juicy ripe peach fruit tissue but not with soft ripe, or dried fruit, or with pieces of integuments or embryo. Fruit tissue from infected plums, apricots, and almonds and wood shields cut from apricot and peach twigs, from which the cambium had been removed, transmitted to peach. The incubation period of the peach mosaic virus in peach varies proportionately with the growth condition at the time of inoculation. Trees inoculated when breaking dormancy and those with 1-inch leaves developed symptoms in 14 and 50 days, respectively. Trees with initial leaves fully expanded usually developed symptoms only in shoots in line with and below the inoculation point. Trees inoculated after June usually developed no symptoms until growth started the following year.

The Mode of Infection and the Incubation Period in the Stem Smut of Grasses, Ustilago hypodytes. GEORGE W. FISCHER. During intensive observation and experimentation on stem smut at Pullman, Washington, it has been discovered that infection occurs in vegetative parts of the host and that the incubation period is at least 2 years and more often 3 years. In planted stands of crested wheatgrass (*Agropyron cristatum*) and other grasses, in no case has stem smut appeared before the third heading of the grass. All blossom and seed inoculations with this smut have given purely negative results. Infection was obtained in crested wheatgrass, slender wheatgrass (*A. trachycaulum*), and Canada wild rye (*Elymus canadensis*), with stem smut from several species of *Agropyron* and *Elymus* only by inoculation of mature plants. These had been clipped back after heading in 1939, sprayed with suspensions of smut spores, and then covered with wet burlap for 48 hours. In 1941, a slight amount of stem smut was noted in some of the slender wheatgrass and crested wheatgrass. In 1942 stem smut was abundant in nearly all rows of slender wheatgrass and crested wheatgrass, and in all of the Canada wild rye. The source of infection of the many species of *Agropyron, Elymus, Hordeum, Poa, Sitanion* and other genera is thought to be in the common heavy infestations of stem smut in quack grass, *Agropyron repens*, in eastern Washington and adjacent Oregon and Idaho.

The Blind-seed Disease of Ryegrass (Lolium spp.) in Oregon. GEORGE W. FISCHER. During 1942 and 1943 considerable difficulty was encountered with low germination of seed of perennial ryegrass, *Lolium perenne*, in sections of the Willamette Valley, Oregon. This reduced germination has now been positively identified with the blind-seed disease (*Phialea temulenta*). Apothecia have been found in abundance on *L. perenne* and *L. temulentum*, and a few on old seeds of *Hordeum gussoneanum*. The conidial stage of the blind-seed fungus was found on *Agrostis exarata* var. *monolepis, Aira caryophyllea, Alopecurus geniculatus, Bromus racemosus, Cynosurus echinatus, Deschampsia caespitosa, Danthonia californica, Festuca elatior, F. myuros, Glyceria borealis, Hordeum gusso-*

neanum, Holcus lanatus, and *Phleum pratense.* A method of qualitative and quantitative detection of blind-seed in current crops of perennial ryegrass and in seed samples is now being worked out. It is a modification of the New Zealand method in that random head samples are chopped and soaked a short time in water, which is then strained through cheesecloth and centrifuged. The residue is then examined microscopically for the presence of the characteristic conidia, the extent of whose presence indicates the severity of the disease in the field represented. No apothecia were found where low-germination (53 per cent) seed was planted in the spring, whereas the same seed, planted in the fall gave an average of 6.8 apothecia per square foot the following spring.

Vegetable Seed-treatment Trials in Western Washington in 1944. C. J. GOULD. Vegetable seed-treatment tests in southern, central, and northern areas of western Washington in the spring of 1944 demonstrated: that materials superior in one area were generally superior in all, despite differences in soil type, rainfall, etc.; that Arasan, Spergon, and Semesan could be safely used on the crops tested by applying an excess of dust to the seed, shaking, and screening off the residue, but that Cuprocide sometimes reduced stands when used in this manner; and that of the new materials tested Arasan was very effective on many vegetable seeds, Dow 5 (tetrachloro-quinone) and Dow 6B (trichloro-phenol) were promising on a few, and U. S. Rubber 604 (dichloronaphtho-quinone) was very good on those on which it was tried. Ten treatments with 5 replications of 200 seeds each were used on each crop at each location. Twenty-six of the 35 tests were statistically significant. The best results were obtained on each crop with the following materials: Peas—Spergon, Semesan, and U.S.R. 604; Beans—Spergon and Semesan; Spinach—Arasan, Cuprocide, U.S.R. 604, and 2 per cent Ceresan; Beets—2 per cent Ceresan and Arasan; Chard—2 per cent Ceresan and Arasan; Squash—Cuprocide and U.S.R. 604; Corn—Arasan and U.S.R. 604; Lettuce—Spergon, Cuprocide, and Semesan; Onion—Arasan and Semesan; and Kale—Semesan and Zinc Oxide.

Leaf Roll of Potato in Washington. LEON K. JONES. Leaf roll has become extremely damaging on potatoes in central Washington since 1938. The disease has spread very rapidly, and infection takes place from the time that plants emerge until the end of the growing season. The virus has been transmitted by grafting and by *Myzus persicae* Sulz., *Myzus pseudosolani* Theob., and *Macrosiphum solanifolii* Ashm., but not by mechanical inoculation methods. The abundant dissemination of the virus during the early season indicates that some other insect, as well as aphids, may also be a carrier of the virus. The yield of potatoes was reduced from normal to about 15 per cent when 100 per cent of planting stock was infected with the virus, to 30 per cent when 25 to 30 per cent of planting tubers were infected, and to 60 per cent of normal when 12 to 15 per cent of planting tubers carried the virus. Early current season infection reduced yields to about 65 per cent of normal and late current season infection caused little or no reduction in yield.

Incidence of Phoma Infection on Sugar-beet Seeds and the Efficiency of Seed Treatments. L. D. LEACH. Among 125 seed lots planted in pasteurized soil in the greenhouse and examined for *Phoma* infection of seedlings, 24 lots were heavily infected. Severity of infection was apparently related to the summer rainfall in the seed-producing areas, being heavy on seed from the Willamette Valley, Oregon, where overhead irrigation is practiced, light to moderate on seed from Medford, Ore., Shasta Valley, Calif., and St. George, Utah, and extremely light on seed from Hemet Valley, Calif. The practice of harvesting two successive seed crops from the same roots often resulted in several times as much *Phoma* infection on the second crop as on the first. Only certain seed lots, planted at low temperature, suffered severe pre-emergence damping-off from seed-borne *Phoma.* In germination trials with controlled soil temperatures, emergence from such lots increased with the temperature up to 25° C., above which only post-emergence infection occurred. Control, but not elimination, of seed-borne infection was obtained by dusting the seed with Ceresan, New Improved Ceresan, Arasan, or dichloro-naphthoquinone. Spergon and Yellow Cuprocide were relatively ineffective. A dip treatment with an ethyl mercury phosphate solution completely eliminated *Phoma* infection from 27 of 31 seed lots tested.

The Application of Vapor Heat as a Practical Means of Disinfecting Seeds. F. P. MCWHORTER and P. W. MILLER. The results of several hundred tests indicate that moist heat supplied by a vapor-heat machine has many advantages over hot water for seed treatment. These are: (1) critical temperature control is unnecessary to insure disinfection and prevent injury to seed; (2) treatment can readily be applied to tons of seed at a time; (3) seeds are only slightly dampened and the same machine removes the excess moisture without rehandling the seeds; and (4) vapor heat can be applied to seeds on moving belts suitable for large scale commercial seed handling. Peas, beets, cabbage, and several grasses will stand temperatures of 140° F. to 150° F. for 90 minutes to 40 minutes, respec-

tively, without significant reduction or retardation of germination. Pathogens belonging to the genera *Macrosporium, Fusarium,* and *Sclerotinia* are consistently killed at much lower temperatures in shorter time. *Phoma* is usually killed and bacterial infestation greatly reduced. Practical control of *Phoma* on beets has been demonstrated. Preliminary tests indicate that *Sclerotinia* as a contaminant of cabbage seed and nematodes in and on grass seeds can be completely eliminated without injury to the seeds.

Transmission Studies with Alfalfa Witches'-broom. J. D. MENZIES. Witches'-broom of alfalfa is graft-transmissible, with symptoms appearing in the stock approximately 2 months after shoot grafting. When root grafting is used, the latent period varies from 5 to 6 months. The disease has been transmitted by shoot grafting to *Medicago lupulina* and *M. hispida.* Grafts of diseased scions to species of *Melilotus* and *Trifolium* all failed to form unions, although many scions remained living for as long as three months. These scions persisted for this length of time by producing roots within the pith tissues of the stock stem. No disease transmission occurred with any of these inter-generic grafts. Witches'-broom could not be transmitted by mechanical sap inoculation nor through the use of dodder. No evidence was obtained of transmission through seed. Insect vector tests have given good evidence that the leafhopper, *Platymoideus acutus* Say., is capable of transmitting witches'-broom. This species occurs throughout the United States and Canada, indicating a danger of future spread of witches'-broom to new areas.

Studies on the Control of Bean Rust. J. A. MILBRATH. Bean rust in Oregon has become of sufficient importance to warrant control only in one area where there is a large acreage of Blue Lake pole beans. Since the uredial stage of the rust has not been found until about 3 weeks after the first appearance of the aecia, probably the telial spores are the only overwintering spores in this area. Large numbers of teliospores can be found adhering to stakes used in the bean yards. Bean yards using old stakes from rusted fields invariably have more rust infection early in the season than those fields or parts of the same field using new stakes. Wire yards usually have less rust than staked yards. When contaminated stakes were placed with beans growing in cages in the greenhouse, numerous pycnial pustules developed in 3 to 4 weeks, while unstaked plants remained free from rust. Dipping contaminated stakes in lime-sulphur 1–10 or in copper sulphate 8–100 prevented infection from the stakes. The commercial dusting sulphur, Kolodust, has greatly reduced rust infection when applied at 7-day intervals, if the dusting program was started before aeciospore formation.

A Disease of Cabbage in Western Oregon Due to Cercosporella albomaculans. P. W. MILLER and F. P. MCWHORTER. During 1943 the foliage of cabbage grown for seed in Coos and Curry counties, Oregon, was attacked by a *Cercosporella.* The disease superficially resembled downy mildew infection, young lesions being black, of dendritic form; the old spots are rounded or rectangular, 10 to 20 mm. in diameter, definitely marginated. Wild turnip, *Brassica oleracea,* occurring as a weed in the plantings was abundantly infected with *Cercospora* (*Cercosporella*) *albomaculans* Ell. et Ev. The species name refers to the white spots characteristic of the fungus on wild turnip. Cross-infections between cabbage and turnips and turnips and cabbage were readily obtained. Conidial characteristics of the fungus on both hosts place it definitely in the former genus *Cercosporella,* a genus given by Ellis and Everhart as second choice when they described the fungus from mustard. The cabbage disease is uncommon and is here interpreted as a case of natural transfer from wild turnip.

Verticillium Wilt of Guayule. HENRY SCHNEIDER. Verticillium wilt has been destructive in field-planted guayule in Kern County and less so in Monterey County, California. It has also been observed in indicator plots in Arizona and Texas. There was considerable variation in the resistance of the different guayule strains to wilt in the field: variety 405 was resistant, variety 109 was very susceptible, and varieties 406 and 593 (the ones commonly planted) were intermediate in susceptibility. In the San Joaquin Valley, Verticillium wilt was active during the spring and fall of 1943 when mean air temperatures were about 70° F. June, 1943, was unusually cool and consequently wilt was active for a longer period than usual. No new infections were noted from July through September when mean air temperatures were about 80° F. Few infections occurred in the fall of 1943 in plots kept dry. In irrigated plots the numbers of infections were inversely proportional to frequency of irrigations. Diseased plants did not recover as well in dry plots as in those that were irrigated.

Copper Sulphate as an Eradicant Spray. C. E. YARWOOD. Solutions containing 0.001 to 0.1 per cent bluestone plus 0.05 per cent spreader applied to infected plants have given fair to excellent eradication of powdery mildews on bean, cucumber, cantaloupe, and rose

without marked host injury in greenhouse and field and have increased the yields of beans, cucumbers, and cantaloupes in the field. The dosage for equivalent eradication decreased with age of infection up to about 8 days, and was less during the day than at night. In greenhouse tests about 6 times as much bluestone was required for 95 per cent eradication when equal lime-Bordeaux (equal quantities of bluestone and of lime) was used as when bluestone without lime was used, but Bordeaux had greater protective action than bluestone. This bluestone spray is considered of value in situations where sulphur dusts or sulphur sprays are unsatisfactory, and powdery mildew is the principal disease to be controlled. As an eradicant spray for bean rust and cucumber downy mildew, bluestone was more effective than Bordeaux, but was effective only up to about 24 hours after inoculation, and is not considered of practical value.

Observations on the Overwintering of Powdery Mildews. C. E. YARWOOD. Of the powdery mildews observed on 175 host species in California, perithecia have been observed on only 44. Heavy mildew infection on a few to many newly opened buds in the spring when most opening buds are healthy, indicates that bud infection may be an important means of overwintering of the powdery mildews on apple, peach, wild rose, lilac, raspberry, grape, oak, plum, cotoneaster, *Photinia serrulata*, *Spirea bumalda*, and *Pyrocantha crenulata*. The occurrence of conidial stages of powdery mildews on rose, cereals, crucifers, Euonymus, Hydrangea and on several weeds during the winter months indicates that these powdery mildews may overwinter as mycelium on the leaves, or as successive conidial generations. Pannose mycelium has been observed on peach and rose twigs during the winter, but one attempt to induce this mycelium to form conidia was unsuccessful. Some powdery mildews have been observed only in greenhouses. The most acceptable manner of overwintering suggested for the powdery mildews of bean and cucurbits is that they overwinter in their conidial stages in southern regions and blow north each season. This same explanation may apply to red clover mildew in Indiana, but there is circumstantial evidence of overwintering as mycelium and as bud infections.

Albino Cherry, a Virus Disease in Southern Oregon. S. M. ZELLER, J. A. MILBRATH, and C. B. CORDY. Albino cherry, apparently a type of buckskin disease, discovered in Ashland in 1937, has now spread as far as Gold Hill, Oregon. Affected Bing or Napoleon trees are usually killed within 3 or 4 years after first symptoms appear, while Lambert, Black Republican, and Montmorency varieties react more slowly. Trees whether on Mazzard or Mahaleb roots behave similarly. The disease is more severe under irrigation than under dry-land culture. All or any portion of a tree may be affected, with die-back a usual symptom of diseased branches. The leaves become a uniform olive brown to golden greenish, with margins rolled upward. Leaves about to shed become chlorotic to orange, with some pinkish tints, especially in a pinnate pattern along the midrib and lateral veins at the base. Late-summer growth of small, green, rosetted leaves is produced from terminal buds on spurs. This characteristic is general on moderately diseased trees although occasionally it is the first symptom observed on otherwise vigorous trees. Fruits remain small and green, turning white even in dark red varieties. Symptoms from inoculations have not proved satisfactory for study since affected trees die within a year after inoculation.

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